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NEWS 1		Web Page URLs for STN Seminar Schedule - N. America
NEWS 2		"Ask CAS" for self-help around the clock
NEWS 3	DEC 18	CA/CAplus pre-1967 chemical substance index entries enhanced with preparation role
NEWS 4	DEC 18	CA/CAplus patent kind codes updated
NEWS 5	DEC 18	MARPAT to CA/CAplus accession number crossover limit increased to 50,000
NEWS 6	DEC 18	MEDLINE updated in preparation for 2007 reload
NEWS 7	DEC 27	CA/CAplus enhanced with more pre-1907 records
NEWS 8	JAN 08	CHEMLIST enhanced with New Zealand Inventory of Chemicals
NEWS 9	JAN 16	CA/CAplus Company Name Thesaurus enhanced and reloaded
NEWS 10	JAN 16	IPC version 2007.01 thesaurus available on STN
NEWS 11	JAN 16	WPIDS/WPINDEX/WPIX enhanced with IPC 8 reclassification data
NEWS 12	JAN 22	CA/CAplus updated with revised CAS roles
NEWS 13	JAN 22	CA/CAplus enhanced with patent applications from India
NEWS 14	JAN 29	PHAR reloaded with new search and display fields
NEWS 15	JAN 29	CAS Registry Number crossover limit increased to 300,000 in multiple databases
NEWS 16	FEB 15	PATDPASPC enhanced with Drug Approval numbers
NEWS 17	FEB 15	RUSSIAPAT enhanced with pre-1994 records
NEWS 18	FEB 23	KOREAPAT enhanced with IPC 8 features and functionality
NEWS 19	FEB 26	MEDLINE reloaded with enhancements
NEWS 20	FEB 26	EMBASE enhanced with Clinical Trial Number field
NEWS 21	FEB 26	TOXCENTER enhanced with reloaded MEDLINE
NEWS 22	FEB 26	IFICDB/IFIPAT/IFIUDB reloaded with enhancements
NEWS 23	FEB 26	CAS Registry Number crossover limit increased from 10,000 to 300,000 in multiple databases
NEWS 24	MAR 15	WPIDS/WPIX enhanced with new FRAGHITSTR display format
NEWS 25	MAR 16	CASREACT coverage extended

NEWS EXPRESS NOVEMBER 10 CURRENT WINDOWS VERSION IS V8.01c, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 25 SEPTEMBER 2006.

NEWS HOURS	STN Operating Hours Plus Help Desk Availability
NEWS LOGIN	Welcome Banner and News Items
NEWS IPC8	For general information regarding STN implementation of IPC 8
NEWS X25	X.25 communication option no longer available

Enter NEWS followed by the item number or name to see news on that specific topic.

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* * * * * * * * * * * * * * * * STN  Columbus  * * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 14:20:14 ON 19 MAR 2007

=> file reg
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.21       0.21

FILE 'REGISTRY' ENTERED AT 14:20:25 ON 19 MAR 2007
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STRUCTURE FILE UPDATES:   16 MAR 2007  HIGHEST RN 926905-73-9
DICTIONARY FILE UPDATES:  16 MAR 2007  HIGHEST RN 926905-73-9

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TSCA INFORMATION NOW CURRENT THROUGH December 2, 2006

  Please note that search-term pricing does apply when
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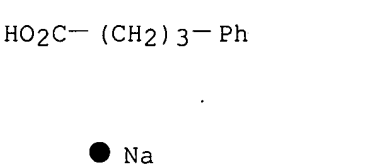
REGISTRY includes numerically searchable data for experimental and
predicted properties as well as tags indicating availability of
experimental property data in the original document.  For information
on property searching in REGISTRY, refer to:

http://www.cas.org/ONLINE/UG/regprops.html

=> s sodium 4-phenylbutyrate
        313317 SODIUM
      18156358 4
           276 PHENYLBUTYRATE
L1           1 SODIUM 4-PHENYLBUTYRATE
                 (SODIUM(W)4(W)PHENYLBUTYRATE)

=> d L1 str cn rn

L1   ANSWER 1 OF 1  REGISTRY  COPYRIGHT 2007 ACS on STN
```




```
CN   Benzenebutanoic acid, sodium salt (1:1)  (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN   Benzenebutanoic acid, sodium salt (9CI)
CN   Butyric acid, 4-phenyl-, sodium salt (8CI)
OTHER NAMES:
CN   Buphenyl
CN   NSC 657802
CN   Sodium γ-phenylbutyrate
CN   Sodium 4-phenylbutyrate
CN   Sodium phenylbutyrate
CN   TriButyrate
```

RN 1716-12-7 REGISTRY

=> file caplus
COST IN U.S. DOLLARS | SINCE FILE ENTRY | TOTAL SESSION
FULL ESTIMATED COST | 17.70 | 17.91

FILE 'CAPLUS' ENTERED AT 14:21:36 ON 19 MAR 2007
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.

FILE COVERS 1907 - 19 Mar 2007 VOL 146 ISS 13
FILE LAST UPDATED: 18 Mar 2007 (20070318/ED)

Effective October 17, 2005, revised CAS Information Use Policies apply.
They are available for your review at:

http://www.cas.org/infopolicy.html

=> s 1716-12-7
REG1stRY INITIATED
Substance data SEARCH and crossover from CAS REGISTRY in progress...
Use DISPLAY HITSTR (or FHITSTR) to directly view retrieved structures.

L3 143 L2

=> s aspartame
4492 ASPARTAME
6 ASPARTAMES
L4 4492 ASPARTAME
(ASPARTAME OR ASPARTAMES)

=> s acesulfame
1108 ACESULFAME
2 ACESULFAMES
L5 1108 ACESULFAME
(ACESULFAME OR ACESULFAMES)

=> s sweetening agent
15817 SWEETENING
22 SWEETENINGS
15826 SWEETENING
(SWEETENING OR SWEETENINGS)
834353 AGENT

```
       1211581 AGENTS
       1703877 AGENT
                 (AGENT OR AGENTS)
L6       12321 SWEETENING AGENT
                 (SWEETENING(W)AGENT)

=> s flavoring agent
         15929 FLAVORING
          1348 FLAVORINGS
         16594 FLAVORING
                 (FLAVORING OR FLAVORINGS)
        834353 AGENT
       1211581 AGENTS
       1703877 AGENT
                 (AGENT OR AGENTS)
L7        2934 FLAVORING AGENT
                 (FLAVORING(W)AGENT)

=> s L3 and L6
L8           2 L3 AND L6

=> s L8 and L7
L9           1 L8 AND L7

=> d L8 1-2 ibib abs

L8   ANSWER 1 OF 2  CAPLUS  COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER:         2007:14088  CAPLUS
DOCUMENT NUMBER:          146:107672
TITLE:                    Process for preparation of liquid dosage form
                          containing sodium 4-phenylbutyrate
INVENTOR(S):              Jobdevairakkam, Christopher Newton; Muthiah, Raja
                          Jeyakumar John
PATENT ASSIGNEE(S):       Navinta LLC, USA
SOURCE:                   U.S. Pat. Appl. Publ., 7pp.
                          CODEN: USXXCO
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.           KIND   DATE        APPLICATION NO.         DATE
     ---------------      ----   --------    --------------------    --------
     US 2007004805         A1    20070104    US 2005-174026          20050701
     WO 2007005633         A2    20070111    WO 2006-US25636         20060629
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
             CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
             GE, GH, GM, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP,
             KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN,
             MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU,
             SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG,
             US, UZ, VC, VN, ZA, ZM, ZW
         RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,
             IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ,
             CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH,
             GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
             KG, KZ, MD, RU, TJ, TM
PRIORITY APPLN. INFO.:                       US 2005-174026      A  20050701
OTHER SOURCE(S):          MARPAT 146:107672
AB   A process for preparing a stable aqueous dosage form of sodium
4-phenylbutyrate,
     including such dosage forms in a highly concentrated solution, as well as
methods
     for making 4-phenylbutyrate and 4-phenylbutyric acid, and for using
```

4-phenylbutyrate. The stable aqueous dosage forms do not freeze at 0°. Sodium 4-phenylbutyrate was transferred to a volumetric flask and about 9 mL water was added and the mixture was then agitated with heating at 70° to dissolve the butyrate. The solution was then cooled to 25° and 0.05 g sodium benzoate and 0.05 g sodium saccharin were added with good mixing. This solution was compounded to 25 mL with water.

L8 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:633292 CAPLUS
DOCUMENT NUMBER: 141:179612
TITLE: Pharmaceutical composition and method for treatment of a urea cycle deficiency or sickle-cell anemia
INVENTOR(S): March, Graham Alan
PATENT ASSIGNEE(S): Special Products Limited, UK
SOURCE: U.S. Pat. Appl. Publ., 11 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
US 2004152784	A1	20040805	US 2003-622891		20030717
PRIORITY APPLN. INFO.:			US 2002-397828P	P	20020723

AB A pharmaceutical composition is disclosed comprising sodium 4-phenylbutyrate, an effective amount of at least one aromatic flavoring agent, and an effective amount of at least one synthetic sweetening agent. Also disclosed is a method of treatment of a urea cycle deficiency or sickle-cell anemia.

=> s L3 and L4
L10 2 L3 AND L4

=> s L3 and L5
L11 1 L3 AND L5

=> s L10 or L11
L12 2 L10 OR L11

=> d 1-2 L12 ibib abs

L12 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2005:244333 CAPLUS
DOCUMENT NUMBER: 143:307
TITLE: Atom, atom-type, and total nonstochastic and stochastic quadratic fingerprints: a promising approach for modeling of antibacterial activity
AUTHOR(S): Marrero-Ponce, Yovani; Medina-Marrero, Ricardo; Torrens, Francisco; Martinez, Yamile; Romero-Zaldivar, Vicente; Castro, Eduardo A.
CORPORATE SOURCE: Department of Pharmacy, Faculty of Chemical-Pharmacy, Central University of Las Villas, Santa Clara, 54830, Cuba
SOURCE: Bioorganic & Medicinal Chemistry (2005), 13(8), 2881-2899
CODEN: BMECEP; ISSN: 0968-0896
PUBLISHER: Elsevier Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The Topol. Mol. Computer Design (TOMOCOMD-CARDD) approach has been introduced for the classification and design of antimicrobial agents using computer-aided mol. design. For this propose, atom, atom-type, and total

quadratic indexes have been generalized to codify chemical structure information. In this sense, stochastic quadratic indexes have been introduced for the description of the mol. structure. These stochastic fingerprints are based on a simple model for the intramol. movement of all valence-bond electrons. In this work, a complete data set containing 1006 antimicrobial agents is collected and presented. Two structure-based antibacterial activity classification models have been generated. The models (including nonstochastic and stochastic indexes) classify correctly more than 90% of 1525 compds. in training sets. These models permit the correct classification of 92.28% and 89.31% of 505 compds. in an external test sets. The approach, also, satisfactorily compares with respect to nine of the most useful models for antimicrobial selection reported to date. Finally, a virtual screening of 87 new compds. reported in the anti-infective field with antibacterial activities is developed showing the ability of the models to identify new leads as antibacterial.

REFERENCE COUNT: 91 THERE ARE 91 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:633292 CAPLUS
DOCUMENT NUMBER: 141:179612
TITLE: Pharmaceutical composition and method for treatment of a urea cycle deficiency or sickle-cell anemia
INVENTOR(S): March, Graham Alan
PATENT ASSIGNEE(S): Special Products Limited, UK
SOURCE: U.S. Pat. Appl. Publ., 11 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
US 2004152784	A1	20040805	US 2003-622891		20030717
PRIORITY APPLN. INFO.:			US 2002-397828P	P	20020723

AB A pharmaceutical composition is disclosed comprising sodium 4-phenylbutyrate, an effective amount of at least one aromatic flavoring agent, and an effective amount of at least one synthetic sweetening agent. Also disclosed is a method of treatment of a urea cycle deficiency or sickle-cell anemia.

=> dup rem L3
PROCESSING COMPLETED FOR L3
L13 143 DUP REM L3 (0 DUPLICATES REMOVED)

=> s L13 and (AY<2004 or PY<2004 or PRY<2004)
L14 143 S L13
4717679 AY<2004
23916714 PY<2004
4199863 PRY<2004
L15 99 L14 AND (AY<2004 OR PY<2004 OR PRY<2004)

=> s armatic flavor
3 ARMATIC
74574 FLAVOR
13438 FLAVORS
80531 FLAVOR
(FLAVOR OR FLAVORS)
L16 0 ARMATIC FLAVOR
(ARMATIC(W)FLAVOR)

=> s aromatic flavor
235153 AROMATIC

```
          9771 AROMATICS
        239664 AROMATIC
                 (AROMATIC OR AROMATICS)
        330117 AROM
         16381 AROMS
        338740 AROM
                 (AROM OR AROMS)
        462064 AROMATIC
                 (AROMATIC OR AROM)
         74574 FLAVOR
         13438 FLAVORS
         80531 FLAVOR
                 (FLAVOR OR FLAVORS)
L17         81 AROMATIC FLAVOR
                 (AROMATIC(W)FLAVOR)

=> s aromatic flavour
        235153 AROMATIC
          9771 AROMATICS
        239664 AROMATIC
                 (AROMATIC OR AROMATICS)
        330117 AROM
         16381 AROMS
        338740 AROM
                 (AROM OR AROMS)
        462064 AROMATIC
                 (AROMATIC OR AROM)
          1158 FLAVOUR
           386 FLAVOURS
          1496 FLAVOUR
                 (FLAVOUR OR FLAVOURS)
L18          0 AROMATIC FLAVOUR
                 (AROMATIC(W)FLAVOUR)

=> s L15 and L17
L19          0 L15 AND L17

=> s L17 and L7
L20          4 L17 AND L7

=> d 1-4 L20 ibib abs

L20  ANSWER 1 OF 4  CAPLUS  COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER:         1989:619131  CAPLUS
DOCUMENT NUMBER:          111:219131
TITLE:                    (E)-4,8-dimethyl-1,3,7-nontriene as a perfume and
                          flavorant
INVENTOR(S):              Maurer, Bruno; Hauser, Arnold
PATENT ASSIGNEE(S):       Firmenich S. A., Switz.
SOURCE:                   Patentschrift (Switz.), 3 pp.
                          CODEN: SWXXAS
DOCUMENT TYPE:            Patent
LANGUAGE:                 French
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.           KIND   DATE        APPLICATION NO.         DATE
     ---------------      ----   --------    --------------------    --------
     CH 668910             A5    19890215    CH 1986-710             19860221
PRIORITY APPLN. INFO.:                       CH 1986-710             19860221
AB   The title compound (I) is prepared as a perfume component and flavorant.  The
     Wittig reaction of (E)-citral with methyltriphenylphosphonium bromide in
     tert-BuOK-containing DMSO gave I.  I is usable in foods, feeds, beverages or
     tobacco.  Addition of 6 g I to 94 g neroli perfume base imparted a green,
```

rich, arom. flavor.

L20 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1976:461668 CAPLUS
DOCUMENT NUMBER: 85:61668
TITLE: Flavoring agent
INVENTOR(S): Winter, Max; Gautschi, Fritz; Flament, Ivon; Stoll, Max; Goldman, Irving M.
PATENT ASSIGNEE(S): Firmenich S. A., Switz.
SOURCE: U.S., 5 pp.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 31
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 3961095	A	19760601	US 1974-482817	19740624
BR 6679143	D0	19730911	BR 1966-179143	19660429
CH 566111	A5	19750915	CH 1970-13417	19660429
GB 1156487	A	19690625	GB 1966-1156487	19660502
NO 134890	B	19760927	NO 1968-108	19680110
NO 134891	B	19760927	NO 1968-1709	19680502
NO 134892	B	19760927	NO 1968-1710	19680502
NO 134893	B	19760927	NO 1968-1711	19680502
NO 134240	B	19760531	NO 1969-5184	19691231
NO 134894	B	19760927	NO 1969-5180	19691231
NO 134895	B	19760927	NO 1969-5181	19691231
NO 134896	B	19760927	NO 1969-5183	19691231
US 3702253	A	19721107	US 1970-70560	19700908
JP 50004736	B	19750224	JP 1971-19574	19710330
US 4303689	A	19811201	US 1972-243850	19720413
DK 139374	B	19790212	DK 1973-5432	19731005
DK 139374	C	19790716		
DK 139454	B	19790226	DK 1973-5428	19731005
DK 139454	C	19790813		
DK 140362	B	19790813	DK 1973-5434	19731005
DK 140362	C	19800114		
DK 139552	C	19790827	DK 1973-5431	19731005
DK 139552	B	19790312		
DK 139605	C	19790903	DK 1973-5426	19731005
DK 139605	B	19790319		
DK 139553	C	19790903	DK 1973-5429	19731005
DK 139553	B	19790312		
DK 139551	C	19790903	DK 1973-5430	19731005
DK 139551	B	19790312		
DK 140243	C	19791203	DK 1973-5427	19731005
DK 140243	B	19790716		
DK 140361	C	19800114	DK 1973-5433	19731005
DK 140361	B	19790813		
US 3900582	A	19750819	US 1974-482818	19740624

PRIORITY APPLN. INFO.:

US 1965-452342	A2	19650430
US 1966-543069	A1	19660418
US 1970-70560	A3	19700908
US 1972-243866	A3	19720413
US 1965-542342	A	19650430
US 1965-543069	A	19650430
DK 1966-2217	A	19660429
NO 1966-162820	A	19660429

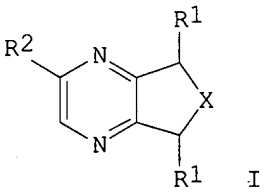
AB The flavor of soluble coffee could be modified by the addition of alkyl naphthalenes. The organoleptic perception of several of these compds., evaluated at 0.05-1.0 g/100 l. 65% sugar solution was tabulated. Thus, α-methyl- [90-12-0], β-methyl- [91-57-6], β-ethyl-

[939-27-5], 1,2-dimethyl- [573-98-8], 1,3-dimethyl- [575-41-7], 1,4-dimethyl- [571-58-4], and 1,5-dimethylnaphthalene [571-61-9] had green-musty, oily-aromatic, oily, aromatic, aromatic, moldy-tarry, and moldy-arom. flavors, resp.

L20 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1976:405688 CAPLUS
DOCUMENT NUMBER: 85:5688
TITLE: Heterocyclic condensed pyrazines
INVENTOR(S): Evers, William J.; Katz, Ira; Theimer, Ernst T.
PATENT ASSIGNEE(S): International Flavors and Fragrances Inc., USA
SOURCE: Ger. Offen., 26 pp. Division of Ger. Offen. 2,117,926.
CODEN: GWXXBX
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 7
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
DE 2166810	A1	19760311	DE 1971-2166810	19710414
DE 2166810	B2	19770908		
US 3647792	A	19720307	US 1970-34583	19700504

PRIORITY APPLN. INFO.: US 1970-34583 A 19700504
GI



I

AB The pyrazines I (X = S, R1 = H, R2 = Me, R1 = R2 = H; X = O, R1 = Me, R2 = H), which have a roasted nut or arom. flavor useful in cosmetics, foods, and tobacco products, were prepared, e.g. by cycloaddn. of 3,4-diaminothiophane with pyruoaldehyde. Also, refluxing 2,3-bis(chloromethyl)pyrazine with NaHS in MeOH for 1.5 hr gave I (X = S, R1 = R2 = H) which in a mixture with acids gave a cheddar cheese flavoring.

L20 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1974:462243 CAPLUS
DOCUMENT NUMBER: 81:62243
TITLE: Effect of hydrocolloids and viscosity on flavor and odor intensities of aromatic flavor compounds
AUTHOR(S): Pangborn, Rose M.; Szczesniak, Alina S.
CORPORATE SOURCE: Dep. Food Sci. Technol., Univ. California, Davis, CA, USA
SOURCE: Journal of Texture Studies (1974), 4(4), 467-82
CODEN: JTXSBU; ISSN: 0022-4901
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The flavor- and odor-modifying effects of low concns. of the hydrocolloids hydroxypropyl cellulose, Na alginate, xanthan, and Na CM-cellulose of low and medium viscosity on the flavoring agents MeCHO, acetophenone, PrCO2H, and Me2S were investigated. Only PrCO2H reduced both phys. and oral viscosity of the hydrocolloids. The addition of hydrocolloids decreased both the odor and flavor intensities of the flavorants. Me2S was affected the most and acetophenone the least. The

flavor of PrCO2H was affected more than the odor. The odor of MeCHO was reduced but the flavor was enhanced.

```
=> s L3 and L7
L21          1 L3 AND L7

=> d L21 ibib abs

L21  ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER:         2004:633292  CAPLUS
DOCUMENT NUMBER:          141:179612
TITLE:                    Pharmaceutical composition and method for treatment of
                          a urea cycle deficiency or sickle-cell anemia
INVENTOR(S):              March, Graham Alan
PATENT ASSIGNEE(S):       Special Products Limited, UK
SOURCE:                   U.S. Pat. Appl. Publ., 11 pp.
                          CODEN: USXXCO
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.          KIND   DATE        APPLICATION NO.         DATE
     ---------------     ----   --------    --------------------    --------
     US 2004152784        A1    20040805    US 2003-622891          20030717
PRIORITY APPLN. INFO.:                      US 2002-397828P     P   20020723
AB   A pharmaceutical composition is disclosed comprising sodium 4-phenylbutyrate,
     an effective amount of at least one aromatic flavoring agent
     , and an effective amount of at least one synthetic sweetening agent.  Also
     disclosed is a method of treatment of a urea cycle deficiency or
     sickle-cell anemia.

=> s strawberry
          7841 STRAWBERRY
          3754 STRAWBERRIES
L22       9082 STRAWBERRY
                 (STRAWBERRY OR STRAWBERRIES)

=> s L3 and L22
L23          0 L3 AND L22

=> s fruit flavor
        104377 FRUIT
         45199 FRUITS
        121858 FRUIT
                 (FRUIT OR FRUITS)
         74574 FLAVOR
         13438 FLAVORS
         80531 FLAVOR
                 (FLAVOR OR FLAVORS)
L24        710 FRUIT FLAVOR
                 (FRUIT(W)FLAVOR)

=> s L3 and L24
L25          1 L3 AND L24

=> d 1 L25 ibib abs

L25  ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER:         2004:633292  CAPLUS
DOCUMENT NUMBER:          141:179612
TITLE:                    Pharmaceutical composition and method for treatment of
```

a urea cycle deficiency or sickle-cell anemia
INVENTOR(S): March, Graham Alan
PATENT ASSIGNEE(S): Special Products Limited, UK
SOURCE: U.S. Pat. Appl. Publ., 11 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
US 2004152784	A1	20040805	US 2003-622891		20030717
PRIORITY APPLN. INFO.:			US 2002-397828P	P	20020723

AB A pharmaceutical composition is disclosed comprising sodium 4-phenylbutyrate, an effective amount of at least one aromatic flavoring agent, and an effective amount of at least one synthetic sweetening agent. Also disclosed is a method of treatment of a urea cycle deficiency or sickle-cell anemia.

=> file medline embase biosis

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	78.71	97.54

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-7.80	-7.80

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FILE 'BIOSIS' ENTERED AT 14:37:15 ON 19 MAR 2007

=> s sodium 4-phenylbutyrate
L26 92 SODIUM 4-PHENYLBUTYRATE

=> s BUPHENYL
L27 20 BUPHENYL

=> s AMMONAPS
L28 17 AMMONAPS

=> dup rem L26
PROCESSING COMPLETED FOR L26
L29 47 DUP REM L26 (45 DUPLICATES REMOVED)

=> dup rem L26
PROCESSING COMPLETED FOR L26
L30 47 DUP REM L26 (45 DUPLICATES REMOVED)

=> dup rem L27
PROCESSING COMPLETED FOR L27
L31 17 DUP REM L27 (3 DUPLICATES REMOVED)

=> dup rem L28
PROCESSING COMPLETED FOR L28
L32 15 DUP REM L28 (2 DUPLICATES REMOVED)

=> s L31 or L32
L33 32 L31 OR L32

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=> dup rem L33
PROCESSING COMPLETED FOR L33
L34          32 DUP REM L33 (0 DUPLICATES REMOVED)

=> s L29 or L34
L35          75 L29 OR L34

=> dup rem L35
PROCESSING COMPLETED FOR L35
L36          75 DUP REM L35 (0 DUPLICATES REMOVED)

=> s L36 and aspatame
L37           0 L36 AND ASPATAME

=> s L36 and aspartame
L38           0 L36 AND ASPARTAME

=> s L36 and flavoring
L39           0 L36 AND FLAVORING

=> s L36 and sweetening agent
L40           0 L36 AND SWEETENING AGENT

=> s povidone
L41       16642 POVIDONE

=> s L36 and L41
L42           0 L36 AND L41

=> d L31 1-5 ibib abs

L31  ANSWER 1 OF 17  EMBASE  COPYRIGHT (c) 2007 Elsevier B.V. All rights
     reserved on STN
ACCESSION NUMBER:    2006290797  EMBASE
TITLE:               Mutation specific therapy in CF.
AUTHOR:              Kerem E.
CORPORATE SOURCE:    E. Kerem, Department of Pediatrics, Cystic Fibrosis Center,
                     Hadassah University Hospital, Mount Scopus, Jerusalem,
                     Israel. ek@cc.huji.ac.il
SOURCE:              Paediatric Respiratory Reviews, (2006) Vol. 7, No. SUPPL.
                     1, pp. S166-S169. .
                     Refs: 18
                     ISSN: 1526-0542  E-ISSN: 1526-0550  CODEN: PRRAEZ
PUBLISHER IDENT.:    S 1526-0542(06)00229-6
COUNTRY:             United Kingdom
DOCUMENT TYPE:       Journal; General Review
FILE SEGMENT:        005     General Pathology and Pathological Anatomy
                     015     Chest Diseases, Thoracic Surgery and Tuberculosis
                     022     Human Genetics
                     029     Clinical Biochemistry
                     030     Pharmacology
                     037     Drug Literature Index
LANGUAGE:            English
SUMMARY LANGUAGE:    English
ENTRY DATE:          Entered STN: 26 Jul 2006
                     Last Updated on STN: 26 Jul 2006
AB   CFTR mutations cause defects of CFTR protein production and function by
     different molecular mechanisms.  The mutations can be classified according
     to the mechanisms by which mutations disrupt CFTR function.  This
     understanding of the different molecular mechanism of CFTR dysfunction
     provides the scientific basis for development of targeted drugs for
     mutation specific therapy of CF.  Class I mutations are nonsense mutations
     that result in the presence of premature stop codon that leads to the
```

production of unstable mRNA or the release from the ribosome of a short truncated protein that is not functional. The aminoglycoside antibiotics can suppress premature termination codons by disrupting translational fidelity and allowing the incorporation of an amino acid, thus permitting translation to continue to the normal termination of the transcript. Class II mutations cause impairment of CFTR processing and folding in the Golgi. As a result the mutant CFTR is retained in the ER and eventually targeted for degradation by the quality control mechanisms. Chemical and molecular chaperons such as Sodium-4-phenylbutyrate can stabilize protein structure, and allow it to escape from degradation in the ER and be transported to the cell membrane. Class III mutations disrupt the function of the regulatory domain. CFTR is resistant to phosphorylation or ATP binding. CFTR activators such as alkylxanthines (CPX) and the flavonoid genistein can overcome the affected ATP binding through direct binding to a nucleotide binding fold. In patients carrying class IV mutations, phosphorylation of CFTR results in reduced chloride transport. Increases in the overall cell surface content of these mutants might overcome the relative reduction in conductance. Alternatively restoring native chloride pore characteristics pharmacologically might be effective. Activators of CFTR at the plasma membrane may function by promoting CFTR phosphorylation, by blocking CFTR dephosphorylation, by interacting directly with CFTR, and/or by modulation of CFTR protein-protein interactions. Class V mutations affect the spicing machinery and generate both aberrantly and correctly spliced transcripts, the level of which vary among different patients and among different organs of the same patient. Splicing factors that promote exon inclusion or factors that promote exon skipping can promote increase of correctly spliced transcripts, depending on the molecular defect. Inconsistent results were reported regarding the required level of corrected or mutated CFTR that has to be reached in order to achieve normal function.

L31 ANSWER 2 OF 17 EMBASE

ACCESSION NUMBER: 2004445318 EMBASE

TITLE: Pharmacological interventions for the correction of ion transport defect in cystic fibrosis.

AUTHOR: Becq F.; Mettey Y.

CORPORATE SOURCE: F. Becq, Inst. de Physiol. Biologie et Cell., CNRS UMR 6187, Universite de Poitiers, 40 Avenue du Recteur Pineau, 86022 Poitiers, France. frederic.becq@univ-poitiers.fr

SOURCE: Expert Opinion on Therapeutic Patents, (2004) Vol. 14, No. 10, pp. 1465-1483. .
Refs: 99
ISSN: 1354-3776 CODEN: EOTPEG

COUNTRY: United Kingdom

DOCUMENT TYPE: Journal; General Review

FILE SEGMENT: 005 General Pathology and Pathological Anatomy
029 Clinical Biochemistry
030 Pharmacology
037 Drug Literature Index
048 Gastroenterology

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 4 Nov 2004
Last Updated on STN: 4 Nov 2004

AB The cystic fibrosis transmembrane conductance regulator (CFTR) is a cAMP-activated and ATP-gated Cl(-) channel expressed in the apical plasma membrane of epithelial cells in the airways, digestive and reproductive tracts. Cystic fibrosis (CF) caused by mutations in the CFTR gene is characterised by chronic airway obstructions and infections, pancreatic failure, male infertility and elevated levels of salt in sweat. A pharmacological therapy would help to restore the defective transepithelial Cl(-) transport observed in CF cells. Therefore,

searching for potent and specific small molecules or peptides able to stimulate transepithelial Cl(-) transport through direct interaction with CFTR or via CFTR-independent mechanisms has become a crucial end point in the field. With the growing understanding of the pharmacology of CFTR activity and processing, a number of academic investigators and biopharmaceutical companies have developed high-throughput screening assays, and reported active seeking of CFTR activators or modulators of airway functions in order to treat CF. This article provides an updated overview of the new emerging molecules and discusses the corresponding patent literature. 2004 .COPYRGT. Ashley Publications Ltd.

L31 ANSWER 3 OF 17 EMBASE

ACCESSION NUMBER: 2004447343 EMBASE
TITLE: Toward the pharmacogenomics of cystic fibrosis - An update.
AUTHOR: Sangiuolo F.; D'Apice M.R.; Gambardella S.; Di Daniele N.; Novelli G.
CORPORATE SOURCE: G. Novelli, Dept. of Biopathology/Diagn. Imaging, Tor Vergata University, via Montpellier 1, 00133 Rome, Italy. novelli@med.uniroma2.it
SOURCE: Pharmacogenomics, (2004) Vol. 5, No. 7, pp. 861-878. .
Refs: 173
ISSN: 1462-2416 CODEN: PARMFL
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
015 Chest Diseases, Thoracic Surgery and Tuberculosis
022 Human Genetics
030 Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 4 Nov 2004
Last Updated on STN: 4 Nov 2004

AB Cystic fibrosis (CF) is the most common autosomal recessive disorder in Caucasians, with a frequency of .apprx. 1 in 3000 live births. The mutated gene is a defective chloride channel in epithelial cells, named cystic fibrosis transmembrane conductance regulator (CFTR). Several different protocols for the scanning of the entire gene have aided molecular diagnosis and improved our understanding of the disorder's pathophysiology, but also showed the disease's complexity. Therefore, CF phenotype remains difficult to predict from CFTR mutation data alone: several studies have suggested that additional genes could modulate its clinical outcome. Gene replacement therapy is still far from being used in patients with CF, mostly due to the difficulties with targeting the appropriate cells. In this review, we summarize recent advances, both in the pharmacological and gene therapy field, aimed for the treatment of the disease. 2004 .COPYRGT. Future Medicine Ltd.

L31 ANSWER 4 OF 17 EMBASE

ACCESSION NUMBER: 2003508272 EMBASE
TITLE: Emerging drugs for renal failure.
AUTHOR: Chatterjee P.K.; Thiemermann C.
CORPORATE SOURCE: Dr. P.K. Chatterjee, Department of Pharmacology, Sch. of Pharm. and Biomol. Sciences, University of Brighton, Moulsecoomb, Brighton, BN2 4GJ, United Kingdom. p.k.chatterjee@brighton.ac.uk
SOURCE: Expert Opinion on Emerging Drugs, (2003) Vol. 8, No. 2, pp. 389-435. .
Refs: 374
ISSN: 1472-8214 CODEN: EOEDA3
COUNTRY: United Kingdom

DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 028 Urology and Nephrology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 22 Jan 2004
Last Updated on STN: 22 Jan 2004

AB Renal failure involves a significant impairment of the essential functions of the kidney, which can be either acute with sudden and rapid onset (acute renal failure [ARF]) or chronic with gradual onset (chronic renal failure [CRF]). ARF, if detected early, may be halted or reversed, whereas CRF is generally irreversible. Without treatment or intervention, both forms of renal failure lead to end stage renal failure (ESRF) or end stage renal disease (ESRD), requiring renal replacement therapy (RRT) in the form of dialysis or renal transplantation for survival. However, provision of RRT requires expert teams working in specialised units, making therapy of patients with renal failure expensive; furthermore, RRT is complex, with its own complications. Although pharmacological interventions have shown promise in experimental models, these have not been as successful in the clinical setting (e.g., administration of atrial natriuretic peptide, low-dose dopamine). At present, drugs are administered during CRF to either reduce one of the many risk factors of CRF (e.g., angiotensin-converting enzyme inhibitors, statins) or to deal with the consequences of CRF (e.g., erythropoietin, calcitriol). Recent evidence suggests that some of these interventions may provide further direct beneficial effects via reduction of renal inflammation. Although these interventions have greatly improved the prospects for patients suffering ESRF, the development of novel drugs and therapies with which to reduce the consequences of renal failure and ESRD remain topics of great interest. This article reviews the therapies available for the prevention and management of renal failure in adults and describes, in detail, emerging drugs and novel interventions that may soon become available for the treatment or prevention of ESRF.

L31 ANSWER 5 OF 17 EMBASE
ACCESSION NUMBER: 2003304173 EMBASE
TITLE: Differentiation therapy.
AUTHOR: Spira A.I.; Carducci M.A.
CORPORATE SOURCE: A.I. Spira, Sidney Kimmel Comprehen. Can. Ctr., 1650 Orleans Street, Baltimore, MD 21231, United States. spiraal@jhmi.edu
SOURCE: Current Opinion in Pharmacology, (2003) Vol. 3, No. 4, pp. 338-343. .
Refs: 56
ISSN: 1471-4892 CODEN: COPUBK
PUBLISHER IDENT.: S 1471-4892(03)00081-X
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 016 Cancer
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 14 Aug 2003
Last Updated on STN: 14 Aug 2003

AB Differentiation therapy is an area of oncology that is in its infancy. Theoretically, the concept of differentiation therapy involves turning a cancer cell 'off' biologically and reverting to a more 'benign' phenotype. Many agents have been studied over the past few years, with many already either in use clinically or showing future promise.

=> d L31 13-17 ibib abs

L31 ANSWER 13 OF 17 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1998310621 EMBASE
TITLE: Therapies directed at the basic defect in cystic fibrosis.
AUTHOR: Zeitlin P.L.
CORPORATE SOURCE: Dr. P.L. Zeitlin, Park 316, Johns Hopkins Hospital, 600 N. Wolfe St., Baltimore, MD 21287, United States
SOURCE: Clinics in Chest Medicine, (1998) Vol. 19, No. 3, pp. 515-525. .
Refs: 84
ISSN: 0272-5231 CODEN: CCHMDA
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 015 Chest Diseases, Thoracic Surgery and Tuberculosis
022 Human Genetics
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 15 Oct 1998
Last Updated on STN: 15 Oct 1998
AB There are over 600 unique mutations in the cystic fibrosis (CF) gene that can be classified in five general categories with respect to specific defect. Through basic research into the genetic and physiologic consequences of these mutations, it has become possible to design genotype-specific therapeutic strategies. New pharmaceutical agents are under development for the rescue of defective cystic fibrosis transmembrane conductance regulator mRNA or protein. Some of these compounds are undergoing study in CF patients in Phase I clinical trials. This article evaluates the current research directed at translating a basic molecular understanding of the disease into innovative new treatments.

L31 ANSWER 14 OF 17 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 1998135729 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9476862
TITLE: A pilot clinical trial of oral sodium 4-phenylbutyrate (Buphenyl) in deltaF508-homozygous cystic fibrosis patients: partial restoration of nasal epithelial CFTR function.
AUTHOR: Rubenstein R C; Zeitlin P L
CORPORATE SOURCE: Eudowood Division of Pediatric Respiratory Sciences, The Johns Hopkins Medical Institutions, Baltimore, Maryland 21287, USA.. rrubenst@welchlink.welch.jhu.edu
CONTRACT NUMBER: P01 HL51811 (NHLBI)
RR00052 (NCRR)
SOURCE: American journal of respiratory and critical care medicine, (1998 Feb) Vol. 157, No. 2, pp. 484-90.
Journal code: 9421642. ISSN: 1073-449X.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199803
ENTRY DATE: Entered STN: 19 Mar 1998
Last Updated on STN: 19 Mar 1998
Entered Medline: 12 Mar 1998
AB Sodium 4-phenylbutyrate (Buphenyl, 4PBA) is a new FDA approved drug for management of urea cycle disorders. We have previously presented

data suggesting that 4PBA, at clinically achievable concentrations, induces CFTR channel function on the plasma membrane of deltaF508-expressing cystic fibrosis (CF) airway epithelial cells in vitro (Rubenstein, R. C., and P. L. Zeitlin, 1997. J. Clin. Invest. 100:2457-2463). We hypothesized that 4PBA would induce epithelial CFTR function in vivo in individuals homozygous for deltaF508-CFTR. A randomized, double-blind, placebo-controlled trial in 18 deltaF508-homozygous patients with CF was performed with the maximum approved adult dose of 4PBA, 19 grams p.o. divided t.i.d., given for 1 wk. Nasal potential difference (NPD) response patterns and sweat chloride concentrations were determined before and after study drug treatment, and 4PBA and metabolites were assayed in plasma and urine at the end of study drug treatment. Subjects in the 4PBA group demonstrated small, but statistically significant improvements of the NPD response to perfusion of an isoproterenol/amiloride/chloride-free solution; this measure reflects epithelial CFTR function and is highly discriminatory between patients with and without CF. Subjects who had received 4PBA did not demonstrate significantly reduced sweat chloride concentrations or alterations in the amiloride-sensitive NPD. Side effects due to drug therapy were minimal and comparable in the two groups. These data are consistent with 4PBA therapy inducing CFTR function in the nasal epithelia of deltaF508-homozygous CF patients.

L31 ANSWER 15 OF 17 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 1997:24753 BIOSIS
DOCUMENT NUMBER: PREV199799323956
TITLE: Sodium phenylbutyrate for urea cycle enzyme deficiencies.
AUTHOR(S): Anonymous
SOURCE: Medical Letter (New Rochelle), (1996) Vol. 38, No. 988, pp. 105-106.
ISSN: 0025-732X.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 15 Jan 1997
Last Updated on STN: 11 Feb 1997

AB Urea cycle disorders, caused by a deficiency of the hepatic enzymes carbamyl phosphate synthetase, ornithine transcarbamylase, or argininosuccinic acid synthetase, are rare, occurring in only 1 out of 10,000 births. The orphan drug sodium phenylbutyrate, manufactured by Ucyclyd Pharma under the trade name Buphenyl, has recently been marketed for the treatment of these disorders. Administered in conjunction with protein restriction and appropriate amino acid supplements, sodium phenylbutyrate can prolong life and preserve cognitive function.

L31 ANSWER 16 OF 17 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 88037312 EMBASE
DOCUMENT NUMBER: 1988037312
TITLE: Microbial transformation of technical mixtures of polychlorinated biphenyls (PCB) by the fungus Aspergillus niger.
AUTHOR: Dmochewitz S.; Ballschmiter K.
CORPORATE SOURCE: Department of Analytical Chemistry, University of Ulm, D-7900 Ulm-Donau, Germany
SOURCE: Chemosphere, (1988) Vol. 17, No. 1, pp. 111-121. .
ISSN: 0045-6535 CODEN: CMSHAF
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal
FILE SEGMENT: 004 Microbiology
046 Environmental Health and Pollution Control
052 Toxicology
LANGUAGE: English

SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 11 Dec 1991
Last Updated on STN: 11 Dec 1991

AB The fungus Aspergillus niger was tested in a replacement culture technique on its ability to transform technical mixtures of polychlorinated buphenyls such as Clophen A 30, A 50 and A 60. A, Niger was selected, because it is widespread in the environment and is discussed as microbial model of mammalian aromatic hydroxylation. The results demonstrate, that A. niger is capable of metabolizing lower chlorinated PCB mixtures (PCB 42% chlorine), whereas no changes of the composition of PCB with higher chlorination levels (PCB 54% and 60% chlorine) could be observed. Substitution in the 4- or 2,5(3,6)-position, respectively, favor the persistence of PCB congeners to the biotransformation by A. niger. A relative order of persistence can be derived for the different congeners found in PCB 42% chlorine.

L31 ANSWER 17 OF 17 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 76175011 EMBASE
DOCUMENT NUMBER: 1976175011
TITLE: Drug metabolism by microsomes from extrahepatic organs of rat and rabbit prepared by calcium aggregation.
AUTHOR: Litterst C.L.; Mimnaugh E.G.; Reagan R.L.; Gram T.E.
CORPORATE SOURCE: Lab. Toxicol., Nat. Cancer Inst., NIH, Bethesda, Md. 20014, United States
SOURCE: Life Sciences, (1975) Vol. 17, No. 5, pp. 813-818. .
CODEN: LIFSAK
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
004 Microbiology
030 Pharmacology
048 Gastroenterology
LANGUAGE: English

AB Microsomes were prepared from liver, lung and kidney of rats and rabbits using a Ca+2 aggregation method. Microsomal protein yield from the lung of both species was higher by this method of preparation as compared with ultracentrifugation samples. Specific activities of rat and rabbit pulmonary p-chloro-N-methylaniline (CMA) demethylase, buphenyl 4-hydroxylase and rat pulmonary TNPH cytochrome c reductase also were decreased. Specific activities of rabbit hepatic TPNH cytochrome c reductase, CMA N-demethylase, UDP-glucuronyltransferase and biphenyl hydroxylase were decreased by calcium aggregation. Renal enzyme activities were unchanged by this method of preparation. These data indicate an apparent species and organ difference in microsomal enzyme response to calcium aggregation.

=> d 6-12 L31 ibib abs

L31 ANSWER 6 OF 17 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 2003:470204 BIOSIS
DOCUMENT NUMBER: PREV200300470204
TITLE: Effect of sodium phenylacetate/benzoate on nitrogen flux and branched chain amino acid metabolism in urea cycle patients and control subjects.
AUTHOR(S): Scaglia, F. [Reprint Author]; O'Brien, W. E. [Reprint Author]; Henry, J. [Reprint Author]; Rosenberger, J. [Reprint Author]; Reeds, P. [Reprint Author]; Lee, B. [Reprint Author]
CORPORATE SOURCE: Department of Molecular and Human Genetics, Children's Nutrition Research Center, and Texas Children's Hospital, Baylor College of Medicine, Houston, TX, USA
SOURCE: Journal of Inherited Metabolic Disease, (September 2003) Vol. 26, No. Supplement 2, pp. 69. print.

Meeting Info.: IX International Congress on Inborn Errors of Metabolism. Brisbane, Australia. September 02-06, 2003. Society for the Study of Inborn Errors of Metabolism.
ISSN: 0141-8955 (ISSN print).
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 8 Oct 2003
Last Updated on STN: 8 Oct 2003

L31 ANSWER 7 OF 17 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2002437189 EMBASE
TITLE: Urea-cycle disorders as a paradigm for inborn errors of hepatocyte metabolism.
AUTHOR: Mian A.; Lee B.
CORPORATE SOURCE: A. Mian, Dept. of Molecular Genetics, Baylor College of Medicine, Houston, TX 77030, United States.
blee@bcm.tmc.edu
SOURCE: Trends in Molecular Medicine, (2002) Vol. 8, No. 12, pp. 583-589. .
Refs: 48
ISSN: 1471-4914 CODEN: TMMRCY
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 029 Clinical Biochemistry
037 Drug Literature Index
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 19 Dec 2002
Last Updated on STN: 19 Dec 2002

AB Urea-cycle disorders (UCDs) are a group of inborn errors of hepatocyte metabolism that are caused by the loss of enzymes involved in the process of transferring nitrogen from ammonia to urea, via the urea cycle (UC). Recent genetic analyses of inherited disorders that present with hyperammonemia demonstrate the function of cellular transporters that regulate the availability of UC intermediates. The regulation of UC intermediates, such as arginine, could have far reaching implications on nitric-oxide synthesis and vascular tone. Hence, each UCD and UC-related disorder constitutes a unique gene-nutrient interaction that is crucial for postnatal homeostasis. Recent advances in the diagnosis and management of UCDs include the application of in vivo metabolic-flux measurements. Cumulative morbidity is still high despite dietary and pharmacological therapies and, hence, both cell and gene therapies are being pursued as possible long-term corrective treatments. Although gene-replacement therapy has suffered recent clinical setbacks, new vector developments offer hope for the treatment of cell-autonomous defects of hepatocyte metabolism.

L31 ANSWER 8 OF 17 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2002352986 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12095312
TITLE: Evidence of CFTR function in cystic fibrosis after systemic administration of 4-phenylbutyrate.
AUTHOR: Zeitlin Pamela L; Diener-West Marie; Rubenstein Ronald C; Boyle Michael P; Lee Carlton K K; Brass-Ernst Lois
CORPORATE SOURCE: Departments of Pediatrics, Johns Hopkins University School of Medicine, Baltimore, Maryland 21287, USA..
pzeitli@jhmi.edu
CONTRACT NUMBER: P01 HL 51811 (NHLBI)
RR00052 (NCRR)
SOURCE: Molecular therapy : the journal of the American Society of Gene Therapy, (2002 Jul) Vol. 6, No. 1, pp. 119-26.

Journal code: 100890581. ISSN: 1525-0016.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CLINICAL TRIAL)
(CLINICAL TRIAL, PHASE I)
(CLINICAL TRIAL, PHASE II)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200301
ENTRY DATE: Entered STN: 4 Jul 2002
Last Updated on STN: 23 Jan 2003
Entered Medline: 22 Jan 2003

AB Most individuals with cystic fibrosis (CF) carry one or two mutations that result in a maturation defect of the full-length protein. One such mutation, deltaF508, results in a mutant membrane glycoprotein that fails to progress to the apical membrane, where the wild-type protein normally functions as a cyclic AMP-regulated chloride channel. 4-Phenylbutyrate (Buphenyl), an orally bioavailable short chain fatty acid, modulates heat shock protein expression and restores maturation of the deltaF508 protein in vitro and in vivo. We performed a randomized, double-blind, placebo-controlled, dose-escalation and safety study of Buphenyl in 19 adults with CF (homozygous deltaF508) to test the hypothesis that Buphenyl would be safe, well-tolerated, and associated with an increase in chloride transport in nasal epithelia. Three dose levels (20, 30, or 40 g divided t.i.d.) of drug or placebo were given for 1 week. Serial measurements of chloride transport by nasal potential difference (NPD) testing and metabolic safety testing were performed. A maximum tolerated dose of 20 g was defined based on minimal adverse reactions, the safety profile, and a statistically significant induction of chloride transport that was maximal by day 3. This short-term phase I/II study demonstrates proof of principle that modulation of deltaF508 CFTR biosynthesis and trafficking is a viable therapeutic approach for cystic fibrosis.

L31 ANSWER 9 OF 17 EMBASE
ACCESSION NUMBER: 2003401940 EMBASE
TITLE: Cystic fibrosis: Can epithelial function be restored?.
AUTHOR: Trapp S.
CORPORATE SOURCE: S. Trapp, Royal Free and University College, Medical School, Rowland Hill Street, London NW3 2PF, United Kingdom. s.trapp@rfc.ucl.ac.uk
SOURCE: IDrugs, (2002) Vol. 5, No. 1, pp. 66-76. .
Refs: 71
ISSN: 1369-7056 CODEN: IDRUFN
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 048 Gastroenterology
029 Clinical Biochemistry
022 Human Genetics
015 Chest Diseases, Thoracic Surgery and Tuberculosis
037 Drug Literature Index
004 Microbiology
030 Pharmacology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 23 Oct 2003
Last Updated on STN: 23 Oct 2003

AB The treatment of cystic fibrosis (CF) is still very much concerned with alleviating its symptoms. However, despite the considerable increase in the knowledge of the function of the cystic fibrosis tmnsmembrane

conductance regulator (CFTR) protein, no magic bullet for the treatment of CF is in sight. This review focuses on what is known about the ion transport defect and which pharmacological and molecular approaches have the greatest potential to provide a cure in the future. .COPYRGT. PharmaPress Ltd.

L31 ANSWER 10 OF 17 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2001009799 EMBASE
TITLE: Pharmacological treatment of the ion transport defect in cystic fibrosis.
AUTHOR: Roomans G.M.
CORPORATE SOURCE: G.M. Roomans, Department of Medical Cell Biology, University of Uppsala, Box 571, 75123 Uppsala, Sweden. godfried.roomans@medcellbiol.uu.se
SOURCE: Expert Opinion on Investigational Drugs, (2001) Vol. 10, No. 1, pp. 1-19. .
Refs: 159
ISSN: 1354-3784 CODEN: EOIDER
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 015 Chest Diseases, Thoracic Surgery and Tuberculosis
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 19 Jan 2001
Last Updated on STN: 19 Jan 2001

AB Cystic fibrosis (CF) is a lethal monogenetic disease characterised by impaired water and ion transport over epithelia. The lung pathology is fatal and causes death in 95% of CF patients. The genetic basis of the disease is a mutation in the cystic fibrosis transmembrane conductance regulator (CFTR), a cAMP-regulated chloride channel. The most common mutation, ΔF508, results in a protein that cannot properly be folded in the endoplasmic reticulum, is destroyed and hence does not reach the apical cell membrane. This paper will discuss those pharmacological approaches that are directed at correcting the defect in ion transport. At present, no clinically effective drug is available, although research has defined areas in which progress might be made. These are the following: (1) the drug 4-phenylbutyrate (4PBA) increases the expression of ΔF508-CFTR in the cell membrane, probably by breaking the association between ΔF508-CFTR and a chaperone; (2) a number of xanthines, in particular 8-cyclopentyl-1, 3-dipropylxanthine (CPX), are effective in activating CFTR, presumably by direct binding and also possibly by correcting the trafficking defect; (3) the isoflavone genistein can activate both wild-type and mutant CFTR, probably through direct binding to the channel; (4) purinergic agonists (ATP and UTP) can stimulate chloride secretion via a Ca(2+)-dependent chloride channel and in this way compensate for the defect in CFTR, but stable analogues will be required before this type of treatment has clinical significance; (5) treatment with inhaled amiloride may correct the excessive absorption of Na(+) ions and water by airway epithelial cells that appears connected to the defect in CFTR; although clinical tests have not been very successful so far, amiloride analogues with a longer half-life may give better results. The role of CFTR in bicarbonate secretion has not yet been established with certainty, but correction of the defect in bicarbonate secretion may be important in clinical treatment of the disease. Currently, major efforts are directed at developing a pharmacological treatment of the ion transport defect in CF, but much basic research remains to be done, in particular, with regard to the mechanism by which defective CFTR is removed in the endoplasmic reticulum by the ubiquitin-proteasome pathway, which is a central pathway in protein production and of significance for several other diseases apart from CF.

L31 ANSWER 11 OF 17 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2001039157 EMBASE
TITLE: Alternative pathway therapy for urea cycle disorders: Twenty years later.
AUTHOR: Batshaw M.L.; MacArthur R.B.; Tuchman M.
CORPORATE SOURCE: Dr. M.L. Batshaw, Children's National Medical Center, 111 Michigan Ave. NW, Washington, DC 20010, United States
SOURCE: Journal of Pediatrics, (2001) Vol. 138, No. 1 SUPPL., pp. S46-S55. .
Refs: 52
ISSN: 0022-3476 CODEN: JOPDAB
COUNTRY: United States
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 007 Pediatrics and Pediatric Surgery
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 15 Feb 2001
Last Updated on STN: 15 Feb 2001

AB Alternative pathway therapy is currently an accepted treatment approach for inborn errors of the urea cycle. This involves the long-term use of oral sodium phenylbutyrate, arginine supplements, or both, depending on the specific enzyme deficiency, and treatment of acute hyperammonemic crises with intravenous sodium benzoate/sodium phenylacetate plus arginine. A review of 20 years of experience with this approach illustrates the strenghts and limitations of this treatment. It has clearly decreased the mortality and morbidity from these disorders, but they remain unacceptably high. The medications are generally well tolerated, but severe accidental overdosage has been reported because of the infrequent use of the medication. There is also a difference in their metabolism between newborns and older children that must be addressed in determining dosage. To avoid these complications it is recommended that drug levels in blood be monitored routinely and that very specific treatment protocols and oversight be followed to avoid overdoses. Finally, it must be acknowledged that alternative pathway therapy has limited effectiveness in preventing hyperammonemia and must be combined with effective dietary management. Therefore in children with neonatal-onset disease or in those with very poor metabolic control, liver transplantation should be considered. There should also be the continued search for innovative therapies that may offer a more permanent and complete correction, such as gene therapy.

L31 ANSWER 12 OF 17 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 1999230436 EMBASE
TITLE: Current developments in the treatment of pulmonary disease in patients with cystic fibrosis.
AUTHOR: Shah P.L.
CORPORATE SOURCE: P.L. Shah, Department of Cystic Fibrosis, Royal Brompton Hospital, Imperial College, Sydney Street, London SW3 6NP, United Kingdom
SOURCE: IDrugs, (1999) Vol. 2, No. 7, pp. 694-701. .
Refs: 90
ISSN: 1369-7056 CODEN: IDRUFN
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 015 Chest Diseases, Thoracic Surgery and Tuberculosis
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 27 Jul 1999

Last Updated on STN: 27 Jul 1999

AB Cystic fibrosis is a genetic disease that affects one in 2500 live births. A basic defect in chloride transport leads to impaired clearance of airway secretions and a susceptibility to bacterial infection. Once infection is established there is a vicious cycle that leads to progressive inflammation and infection. Although cystic fibrosis is a multisystem disorder, pulmonary disease is the main cause of morbidity and respiratory failure remains the main cause of death. This review discusses the strategies for treating pulmonary disease in patients with cystic fibrosis and focuses on some of the therapeutic developments.

=> d L32 1-15 ibib abs

L32 ANSWER 1 OF 15 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2006190098 EMBASE

TITLE: Sustained Engraftment and Tissue Enzyme Activity After Liver Cell Transplantation for Argininosuccinate Lyase Deficiency.

AUTHOR: Stephenne X.; Najimi M.; Sibille C.; Nassogne M.; Smets F.; Sokal E.M.

CORPORATE SOURCE: E.M. Sokal, Laboratoire d'hepatologie Pediatrique et Transplantation Cellulaire, Departement GYPE, Service de Pediatrie, Brussels, Belgium. Sokal@pedi.ucl.ac.be

SOURCE: Gastroenterology, (2006) Vol. 130, No. 4, pp. 1317-1323. .
Refs: 35
ISSN: 0016-5085 CODEN: GASTAB

PUBLISHER IDENT.: S 0016-5085(06)00009-6

COUNTRY: United States

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 007 Pediatrics and Pediatric Surgery
037 Drug Literature Index
048 Gastroenterology

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 6 Jun 2006
Last Updated on STN: 6 Jun 2006

AB Background & Aims: Donor cell engraftment with expression of enzyme activity is the goal of liver cell transplantation for inborn errors of liver metabolism with a view to achieving sustained metabolic control. Methods: Sequential hepatic cell transplantations using male and female cells were performed in a 3.5-year-old girl with argininosuccinate lyase deficiency over a period of 5 months. Beside clinical, psychomotor, and metabolic follow-up, engraftment was analyzed in repeated liver biopsies (2.5, 5, 8, and 12 months after first infusion) by fluorescence in situ hybridization for the Y-chromosome and by measurement of tissue enzyme activity. Results: Metabolic control was achieved together with psychomotor catch-up, changing the clinical phenotype from a severe neonatal one to a moderate late-onset type. The child was no longer hospitalized and was able to attend normal school. Sustained engraftment of male donor liver cells was shown in repeated biopsies, reaching 19% at 8 months and 12.5% at the 12-month follow-up. XXYY tetraploid donor cells were mainly detected during the infusion period (2.5- and 5-month biopsies), whereas in the follow-up 8-month and 1-year biopsies, diploid donor cell subpopulations had become dominant. Moreover, argininosuccinate lyase activity, originally absent, became measurable in 2 different biopsy samples at 8 months, reaching 3% of control activity, indicating in situ metabolic effect and supporting the clinical evolution to a moderate form of the disease. Conclusions: Liver cell transplantation can achieve donor cell engraftment in humans in a significant proportion, leading to sustained metabolic and clinical control with psychomotor catch-up. .COPYRGT. 2006 American Gastroenterological Association Institute.

L32 ANSWER 2 OF 15 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 2007:82785 BIOSIS
DOCUMENT NUMBER: PREV200700089312
TITLE: Outcomes among male OTC patients treated with ammonaps (R).
AUTHOR(S): Wuebbels, B. H. [Reprint Author]
CORPORATE SOURCE: Ucyclyd Pharma Inc, Dept Clin Educ, Scottsdale, AZ USA
SOURCE: Journal of Inherited Metabolic Disease, (AUG 2006) Vol. 29, No. Suppl. 1, pp. 129.
Meeting Info.: 10th International Congress of Inborn Errors of Metabolism (ICIEM). Chiba, JAPAN. September 12 -16, 2006.
CODEN: JIMDDP. ISSN: 0141-8955.
DOCUMENT TYPE: Conference; (Meeting)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 31 Jan 2007
Last Updated on STN: 31 Jan 2007

L32 ANSWER 3 OF 15 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2006230683 EMBASE
TITLE: [Pharmacological treatment of congenital metabolic disorders].
TRATAMIENTO FARMACOLOGICO DE LOS TRASTORNOS METABOLICOS CONGENITOS.
AUTHOR: Campino Villegas A.
CORPORATE SOURCE: A. Campino Villegas, Servicio de Farmacia, Hospital de Cruces, Barakaldo. Vizcaya, Spain
SOURCE: Atencion Farmaceutica, (2006) Vol. 8, No. 1, pp. 39-46. .
Refs: 16
ISSN: 1139-7357 CODEN: AFARFP
COUNTRY: Spain
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 003 Endocrinology
022 Human Genetics
037 Drug Literature Index
LANGUAGE: Spanish
SUMMARY LANGUAGE: English; Spanish
ENTRY DATE: Entered STN: 29 May 2006
Last Updated on STN: 29 May 2006
AB Inborn errors of metabolism, or congenital metabolic disorders, are a multiple number of diseases that, in many occasions, are unknown to the hospital pharmacist due to their lower prevalence. However, it is necessary to acquire some knowledge about these disorders and their treatments, because, in some instances, the pharmacological treatment is essential during acute situations. In this last ten years of Medline's review, we collect some of the most frequent congenital metabolic disorders, along with their respective pharmacological treatments and dietary requirements. Moreover, we explain how to obtain the various medications: request for foreign medication or compassionate use.

L32 ANSWER 4 OF 15 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2005064306 EMBASE
TITLE: [Activity of the CHMP].
AKTIVITATEN DES CHMP.
AUTHOR: Throm S.
CORPORATE SOURCE: Dr. S. Throm, VFA - e.V., Geschaftsfuhrer Forsch., E., I., Hausvogteiplatz 13, 10117 Berlin, Germany. s.throm@vfa.de
SOURCE: Pharmazeutische Industrie, (2005) Vol. 67, No. 1, pp. 57-63. .
ISSN: 0031-711X CODEN: PHINAN

COUNTRY: Germany
DOCUMENT TYPE: Journal; (Short Survey)
FILE SEGMENT: 037 Drug Literature Index
039 Pharmacy
LANGUAGE: German
ENTRY DATE: Entered STN: 18 Feb 2005
Last Updated on STN: 18 Feb 2005
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L32 ANSWER 5 OF 15 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2005030961 EMBASE
TITLE: [Orphan drug: Carglumic acid for the treatment of urea cycle disorders].
ORPHAN DRUG: NEU BEI STORUNGEN DES HARNSTOFFZYKLUS: CARGLUMSAURE.
SOURCE: Deutsche Apotheker Zeitung, (6 Jan 2005) Vol. 145, No. 1, pp. 44-46. .
ISSN: 0011-9857 CODEN: DAZEA2
COUNTRY: Germany
DOCUMENT TYPE: Journal; (Short Survey)
FILE SEGMENT: 030 Pharmacology
037 Drug Literature Index
039 Pharmacy
LANGUAGE: German
ENTRY DATE: Entered STN: 27 Jan 2005
Last Updated on STN: 27 Jan 2005
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L32 ANSWER 6 OF 15 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2004159590 EMBASE
TITLE: [Activities of the CPMP].
AKTIVITATEN DES CPMP.
AUTHOR: Throm S.
CORPORATE SOURCE: Dr. S. Throm, VFA - Verband Forschender A. e.V., Geschaftsfuhrer Forschung, Hausvogteiplatz 13, 10117 Berlin, Germany. s.throm@vfa.de
SOURCE: Pharmazeutische Industrie, (2004) Vol. 66, No. 3, pp. 294-297. .
ISSN: 0031-711X CODEN: PHINAN
COUNTRY: Germany
DOCUMENT TYPE: Journal; (Short Survey)
FILE SEGMENT: 006 Internal Medicine
037 Drug Literature Index
039 Pharmacy
LANGUAGE: German
ENTRY DATE: Entered STN: 6 May 2004
Last Updated on STN: 6 May 2004
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L32 ANSWER 7 OF 15 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2003289014 EMBASE
TITLE: [Activities of the CPMP].
AKTIVITATEN DES CPMP.
AUTHOR: Throm S.
CORPORATE SOURCE: Dr. S. Throm, VFA, Geschaftsfuhrer Forsch., E., I., Hausvogteiplatz 13, 10117 Berlin, Germany. s.throm@vfa.de
SOURCE: Pharmazeutische Industrie, (2003) Vol. 65, No. 5, pp. 403-406. .
ISSN: 0031-711X CODEN: PHINAN
COUNTRY: Germany
DOCUMENT TYPE: Journal; (Short Survey)

FILE SEGMENT: 036 Health Policy, Economics and Management
037 Drug Literature Index
039 Pharmacy
LANGUAGE: German
ENTRY DATE: Entered STN: 31 Jul 2003
Last Updated on STN: 31 Jul 2003
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L32 ANSWER 8 OF 15 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2002123760 EMBASE
TITLE: [Activities of the CPMP].
AKTIVITATEN DES CPMP.
AUTHOR: Throm S.
CORPORATE SOURCE: Dr. S. Throm, VFA - Verb. Forsch. Arzneimitt. e.V., Geschaftsfu. Forsch. Entwick. Innov., Hausvogteiplatz 13, 10117 Berlin, Germany. s.throm@vfa.de
SOURCE: Pharmazeutische Industrie, (2002) Vol. 64, No. 3, pp. 234-240. .
ISSN: 0031-711X CODEN: PHINAN
COUNTRY: Germany
DOCUMENT TYPE: Journal; (Short Survey)
FILE SEGMENT: 037 Drug Literature Index
039 Pharmacy
LANGUAGE: German
ENTRY DATE: Entered STN: 25 Apr 2002
Last Updated on STN: 25 Apr 2002
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L32 ANSWER 9 OF 15 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2002235814 EMBASE
TITLE: [News about drugs for children].
NOUVEAUTES EN MATIERE DE MEDICAMENTS DESTINES AUX ENFANTS.
AUTHOR: Autret-Leca E.; Jonville Bera A.P.
CORPORATE SOURCE: E. Autret-Leca, Service de Pharmacologie, CHRU, 37044 Tours Cedex, France
SOURCE: Journal de Pediatrie et de Puericulture, (2002) Vol. 15, No. 4, pp. 219-223. .
ISSN: 0987-7983 CODEN: JPPUF6
COUNTRY: France
DOCUMENT TYPE: Journal; (Short Survey)
FILE SEGMENT: 007 Pediatrics and Pediatric Surgery
037 Drug Literature Index
LANGUAGE: French
ENTRY DATE: Entered STN: 18 Jul 2002
Last Updated on STN: 18 Jul 2002
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L32 ANSWER 10 OF 15 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2006578112 EMBASE
TITLE: [Recent drugs intended for children].
LES MEDICAMENTS RECENTS DESTINES AUX ENFANTS.
AUTHOR: Autret-Leca E.; Jonville Bera A.P.
CORPORATE SOURCE: E. Autret-Leca, Service de Pharmacologie, CHRU de Tours
SOURCE: Actualites Pharmaceutiques, (2002) No. 412, pp. 47-50. .
ISSN: 0515-3700 CODEN: ACPHDD
COUNTRY: France
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 017 Public Health, Social Medicine and Epidemiology
037 Drug Literature Index
LANGUAGE: French
ENTRY DATE: Entered STN: 7 Dec 2006

Last Updated on STN: 7 Dec 2006
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L32 ANSWER 11 OF 15 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2002227685 EMBASE
TITLE: [Sodium phenylbutyrate for the treatment of urea cycle disorders].
NEUE ARZNEISTOFFE: NATRIUMPHENYLBUTYRAT BEI STORUNGEN IM HARNSTOFFZVKLUS.
AUTHOR: Bertsche T.; Schulz M.
CORPORATE SOURCE: T. Bertsche, Zentrum fur Arzneimittelinformation, Pharmazeutische Praxis, Carl-Mannich-Strasse 26, 65760 Eschborn, Germany
SOURCE: Pharmazeutische Zeitung, (20 Jun 2002) Vol. 147, No. 25, pp. 28-34. .
Refs: 6
ISSN: 0031-7136 CODEN: PZSED5
COUNTRY: Germany
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 006 Internal Medicine
029 Clinical Biochemistry
037 Drug Literature Index
LANGUAGE: German
ENTRY DATE: Entered STN: 11 Jul 2002
Last Updated on STN: 11 Jul 2002
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L32 ANSWER 12 OF 15 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2001270711 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11286510
TITLE: Long-term treatment with sodium phenylbutyrate in ornithine transcarbamylase-deficient patients.
AUTHOR: Burlina A B; Ogier H; Korall H; Trefz F K
CORPORATE SOURCE: Department of Paediatrics, University of Padova, Padova, Italy.. burlina@child.pedi.unipd.it
SOURCE: Molecular genetics and metabolism, (2001 Apr) Vol. 72, No. 4, pp. 351-5.
Journal code: 9805456. ISSN: 1096-7192.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200106
ENTRY DATE: Entered STN: 25 Jun 2001
Last Updated on STN: 25 Jun 2001
Entered Medline: 21 Jun 2001
AB Ornithine transcarbamylase deficiency is a very heterogeneous urea cycle disorder resulting in hyperammonemia with various presentations from the neonatal period through adulthood. We performed a retrospective study in nine patients (four male/five female, age at diagnosis ranging from 6 days to 14 years) to evaluate the safety and efficacy of sodium phenylbutyrate (Ammonaps) in long-term treatment. All patients were diagnosed by DNA mutational analysis and/or liver enzyme measurement. They had previously been treated with sodium benzoate (median dose 248 mg/kg/day; range 106-275) and low protein diet (median 0.84 g/kg/day) and were switched to sodium phenylbutyrate (median dose of 352 mg/kg/day) at 8.9 and 4.9 years of age (median) in males and females, respectively. We analyzed clinical and biochemical data and the median follow-up duration was 26 months. During that time, there were no hyperammonemic episodes requiring hospitalization. Median plasma ammonia and glutamine levels were 30 and 902 micromol/L, respectively. Total protein intake could be increased to 0.95 g/kg/day after 18 months. No side effects related to

therapy were observed. Further prospective studies should be performed to define the optimal dosage of sodium phenylbutyrate and the requirements for protein diet at different ages.

L32 ANSWER 13 OF 15 EMBASE
ACCESSION NUMBER: 2001101954 EMBASE
TITLE: [Activities of the CPMP].
AKTIVITATEN DES CPMP.
AUTHOR: Throm S.
CORPORATE SOURCE: Dr. S. Throm, VFA - Verband Forschender, Arzneimittelhersteller e.V., Produktion, Qualitat und Umwelt, Hausvogteiplatz 13, 10117 Berlin, Germany. s.throm@vfa.de
SOURCE: Pharmazeutische Industrie, (2001) Vol. 63, No. 2, pp. 138-145. .
ISSN: 0031-711X CODEN: PHINAN
COUNTRY: Germany
DOCUMENT TYPE: Journal; (Short Survey)
FILE SEGMENT: 037 Drug Literature Index
LANGUAGE: German
ENTRY DATE: Entered STN: 6 Apr 2001
Last Updated on STN: 6 Apr 2001
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L32 ANSWER 14 OF 15 EMBASE
ACCESSION NUMBER: 2001231661 EMBASE
TITLE: [Pharmacon in Merano: Drugs for immediate use in the pharmacy].
PHARMACON MERAN: ZUM SOFORTIGEN GEBRAUCH IN DER OFFIZIN.
AUTHOR: Brunner U.; Gensthaler B.M.; Morck H.
SOURCE: Pharmazeutische Zeitung, (7 Jun 2001) Vol. 146, No. 23, pp. 26-34. .
ISSN: 0031-7136 CODEN: PZSED5
COUNTRY: Germany
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 037 Drug Literature Index
LANGUAGE: German
ENTRY DATE: Entered STN: 19 Jul 2001
Last Updated on STN: 19 Jul 2001
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L32 ANSWER 15 OF 15 BIOSIS
ACCESSION NUMBER: 2001:66585 BIOSIS
DOCUMENT NUMBER: PREV200100066585
TITLE: Long-term treatment with sodium phenylbutyrate in ornithine transcarbamylase (OTC) deficient patients.
AUTHOR(S): Burlina, A. B. [Reprint author]; Ogier, H.; Korall, H.; Trefz, F.
CORPORATE SOURCE: Dept. Paediatrics, University of Padova, Padova, Italy
SOURCE: Journal of Inherited Metabolic Disease, (July, 2000) Vol. 23, No. Supplement 1, pp. 54. print.
Meeting Info.: VIIIth International Conference on Inborn Errors of Metabolism. England, Cambridge, UK. September 13-17, 2000.
CODEN: JIMDDP. ISSN: 0141-8955.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 31 Jan 2001
Last Updated on STN: 12 Feb 2002

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L44          143 S L43
         4166694 AY<2002
        21882372 PY<2002
         3643772 PRY<2002
L45           72 L44 AND (AY<2002 OR PY<2002 OR PRY<2002)

=> d 1-10 L45 ibib abs

L45  ANSWER 1 OF 72  CAPLUS  COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER:         2007:175142  CAPLUS
DOCUMENT NUMBER:          146:244322
TITLE:                    Novel methods of cancer diagnosis and therapy targeted
                          against a cancer stem line
INVENTOR(S):              Bergstein, Ivan
PATENT ASSIGNEE(S):       USA
SOURCE:                   U.S. Pat. Appl. Publ., 46pp., Cont. of U.S. Ser. No.
                          468,286.
                          CODEN: USXXCO
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   2
PATENT INFORMATION:

     PATENT NO.           KIND   DATE        APPLICATION NO.          DATE
     ---------------      ----   --------    --------------------     --------
     US 2007036800         A1    20070215    US 2006-583744           20061018 <--
```

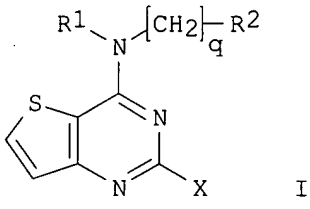
US 6004528 A 19991221 US 1997-933330 19970918 <--
PRIORITY APPLN. INFO.: US 1997-933330 A2 19970918 <--
US 1999-468286 A1 19991220 <--

AB Improved methods for treatment of cancer which involve the targeting of slow-growing, relatively mutationally-spared cancer stem line are provided. These methods are an improvement over previous cancer therapeutic methods because they provide for very early cancer treatment and reduce the likelihood of clin. relapse after treatment.

L45 ANSWER 2 OF 72 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2003:532671 CAPLUS
DOCUMENT NUMBER: 139:101145
TITLE: Preparation of thienopyrimidines as inhibitors of prolylpeptidase, inducers of apoptosis and cancer treatment agents
INVENTOR(S): Dumas, Jacques; Sibley, Robert; Wood, Jill
PATENT ASSIGNEE(S): Bayer Corporation, USA
SOURCE: PCT Int. Appl., 50 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003055890	A1	20030710	WO 2002-US41168	20021220 <--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2002364211	A1	20030715	AU 2002-364211	20021220 <--

PRIORITY APPLN. INFO.: US 2001-343048P P 20011221 <--
WO 2002-US41168 W 20021220
OTHER SOURCE(S): MARPAT 139:101145
GI



I

AB The title compds. [I; X = OR3, NR3R4; R1 = H, alkyl; R2 = (un)substituted cycloalkyl, Ph, (un)saturated 4-8 membered heterocyclyl containing 1-3 heteroatoms selected from O and S; R3 = H, alkyl; R4 = (CH2)mA, (CH2)pOA; A = (un)substituted cycloalkyl, (un)saturated 4-8 membered heterocyclyl containing 1-4 heteroatoms selected from N, O and S, etc.; or NR3R4 = (un)saturated 4-8 membered heterocyclyl containing 0-4 heteroatoms selected from N, O and S; m, p = 0-5; q = 0-1; q + (m or p) = 1-6], useful for the inhibiting the prolylpeptidase, inducing apoptosis and treating cancer, were prepared

E.g., a 3-step synthesis of I [X = (2-thienylmethyl)amino; R1 = H; R2 = 4-(MeO2C)C6H4; q = 1], starting with thieno[3,2-d]pyrimidine-2,4-diol, was given. All exemplified compds. I were found to inhibit prolylpeptidase at or below of 10 μM.

REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

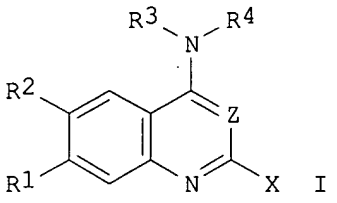
L45 ANSWER 3 OF 72 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2003:532653 CAPLUS
DOCUMENT NUMBER: 139:101144
TITLE: Preparation of quinazolines and quinolines as inhibitors of prolylpeptidase, inducers of apoptosis and cancer treatment agents
INVENTOR(S): Dumas, Jacques; Sibley, Robert; Smith, Roger; Su, Ning; Chen, Yuanwei; Wood, Jill; Guernon, Leatte; Dixon, Julie; Brennan, Catherine; Boyer, Stephen
PATENT ASSIGNEE(S): Bayer Corporation, USA; et al.
SOURCE: PCT Int. Appl., 266 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003055866	A1	20030710	WO 2002-US41176	20021220 <--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2002361846	A1	20030715	AU 2002-361846	20021220 <--

PRIORITY APPLN. INFO.: US 2001-343112P P 20011221 <--
WO 2002-US41176 W 20021220

OTHER SOURCE(S): MARPAT 139:101144
GI



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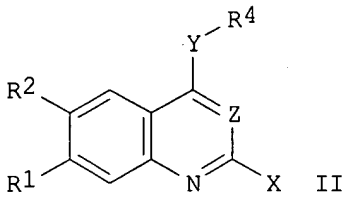


II

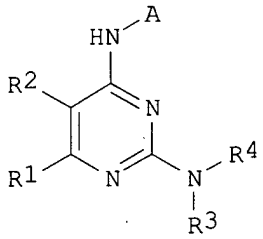
AB The title compds. [I or II; Z = CH, N; Y = O, S; X = OR5, NR5R6; R1, R2 = H, NH2, CN, halo, OH, NO2 (wherein R1 and R2 are both not H); R3 = H, alkyl; R4 = (CH2)yR41 (R41 = (un)substituted alkyl; y = 0-2)], useful for the inhibiting the prolyl peptidase, inducing apoptosis and treating cancer, were prepared Thus, reacting 2,4,6-trichloroquinazoline (preparation given) with Me 4-(aminomethyl)benzoate.HCl in the presence of AcONa in H2O followed by treating the resulting Me 4-{[(2,6-dichloro-4-quinazolinyl)amino]methyl}benzoate with piperidine afforded I [Z = N; X = piperidino; R1 = H; R2 = Cl; R3 = H; R4 = 4-(MeO2C)C6H4CH2]. Most of the exemplified compds. I and II were found to inhibit prolylpeptidase at or

below of 10 μM.
REFERENCE COUNT: 48 THERE ARE 48 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L45 ANSWER 4 OF 72 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2003:532524 CAPLUS
DOCUMENT NUMBER: 139:101141
TITLE: Preparation of 2,4-diaminopyrimidines as inhibitors of prolylpeptidase, inducers of apoptosis and cancer treatment agents
INVENTOR(S): Dumas, Jacques; Dixon, Julie; Sibley, Robert; Wood, Jill
PATENT ASSIGNEE(S): Bayer Corporation, USA
SOURCE: PCT Int. Appl., 47 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003055489	A1	20030710	WO 2002-US41146	20021220 <--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2002367172	A1	20030715	AU 2002-367172	20021220 <--
PRIORITY APPLN. INFO.:			US 2001-343047P P	20011221 <--
			WO 2002-US41146 W	20021220

OTHER SOURCE(S): MARPAT 139:101141
GI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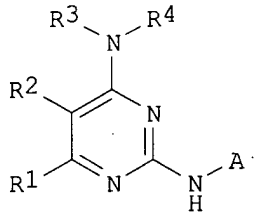
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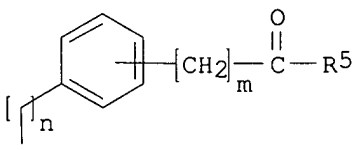
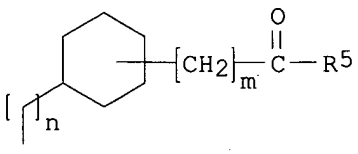

III


IV

AB The title compds. [I or II; R1, R2 = H, halo, OH, etc.; R3 = H; R4 =

(un)substituted alkyl, cycloalkyl, aryl, alkylaryl; or NR3R4 = (un)saturated 4-8 membered heterocyclyl which optionally contains 1-3 addnl. heteroatoms selected from N, O and S; A = III or IV; R5 = OH, OR6, NR8R9; R6 = alkyl, haloalkyl, aryl, haloaryl; R8, R9 = H, alkyl, aryl, etc.; n, m = 0-1], useful for the inhibiting prolylpeptidase, inducing apoptosis and treating cancer, were prepared E.g., a 3-step synthesis of I [A = 4-(HO2C)C6H4CH2; R1 = H; R2 = Me; R3 = H; R4 = 2-thienylmethyl], starting from Me 4-(aminomethyl)benzoate and 2,4-dichloro-5-methylpyrimidine, was given. All exemplified compds. I were found to inhibit prolylpeptidase at or below of 10 μM.

REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L45 ANSWER 5 OF 72 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2003:335262 CAPLUS
DOCUMENT NUMBER: 138:349698
TITLE: Screening system for modulators of gene HER2 (neu/ErbB2) transcription, HER2 modulators identified thereby, and methods involving HER2 SNPs
INVENTOR(S): Benz, Christopher C.
PATENT ASSIGNEE(S): Buck Institute for Age Research, USA
SOURCE: PCT Int. Appl., 103 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 2003035843	A2	20030501	WO 2002-US34288		20021025 <--
WO 2003035843	A3	20040826			
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW					
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG					
US 2005123896	A1	20050609	US 2003-493141		20021025 <--
PRIORITY APPLN. INFO.:			US 2001-346262P	P	20011025 <--
			US 2001-335290P	P	20011130 <--
			US 2002-374161P	P	20020417
			WO 2002-US34288	W	20021025

AB This invention pertains to the development of a screening system to identify (screen for) gene HER2 (neu/ErbB2) promoter silencing agents. Such agents are expected to be of therapeutic value in the treatment of cancers characterized by HER2 amplification/upregulation. In addition, this invention pertains to the discovery that histone deacetylase (HDAC) inhibitors like sodium butyrate and trichostatin A (TSA), in a time and dose dependent fashion can silence genomically integrated and/or amplified/overexpressing promoters, such as that driving the HER2 (neu/ErbB2) oncogene, resulting in inhibition of gene products including transcripts and protein, and subsequent production of tumor/cell growth inhibition, apoptosis and/or differentiation. In another embodiment, this invention provides novel single nucleotide polymorphisms (SNPs) associated with the coding region of the HER2 proto-oncogene. The SNPs are indicators for altered risk, for developing ErbB2-pos. cancer in a mammal.

L45 ANSWER 6 OF 72 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2003:201472 CAPLUS

DOCUMENT NUMBER: 138:210369
TITLE: Prolonged-release forms pharmaceutical dosage forms
INVENTOR(S): Truog, Peter
PATENT ASSIGNEE(S): Lunamed AG, Switz.
SOURCE: Eur. Pat. Appl., 8 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 1291015	A1	20030312	EP 2001-810865	20010910 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
CA 2459165	A1	20030320	CA 2002-2459165	20020904 <--
WO 2003022253	A1	20030320	WO 2002-CH486	20020904 <--
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RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1427396	A1	20040616	EP 2002-754105	20020904 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				
CN 1553799	A	20041208	CN 2002-817630	20020904 <--
JP 2005508901	T	20050407	JP 2003-526383	20020904 <--
US 2004180962	A1	20040916	US 2004-488276	20040226 <--

PRIORITY APPLN. INFO.: EP 2001-810865 A 20010910 <--
WO 2002-CH486 W 20020904

AB A pharmaceutical unit dosage form comprises a therapeutically ED of a 4-phenylbutyric acid salt having prolonged release of the active ingredient, being suitable for alleviating and curing various diseases upon once or twice daily oral administration. A method for the preparation of the pharmaceutical formulation and the use thereof for the treatment of benign prostate hyperplasia, cancer, leukemias, cystic fibrosis, AIDS, kidney and liver diseases, thalassemia and urea cycle disorders after twice-daily oral administration of the formulation to a patient is also disclosed. A mixture of 6000.0 g sodium 4-phenylbutyrate, 6280.0 g lactose monohydrate, 3500.0 g Methocel K100M, and 750.0 g Avicel PH102, is wetted with 4000.0 g with water, and dried in cold air for 18 h. The mixture is forced through a sieve and dried again for 10 h with air of 40°. A mixture of 240.0 g talcum and 30.0 g magnesium stearate is admixed for 20 min and the mixture is pressed into tablets of 0.70 g each. The cores are provided with a film coating by using a colloidal dispersion containing 7850 g iso-PrOH, 3360 g Eudragit L12.5, 66 g di-Bu phthalate, 18.0 g Miglyol-812 and 56 g PEG-400. The film-coated tablets are dried in a circulating air drying cabinet for at least 4 h at 35°.

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L45 ANSWER 7 OF 72 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2003:89718 CAPLUS
DOCUMENT NUMBER: 138:112406
TITLE: Percutaneous absorption type anticancer medicine to suppress hyperplasia and untoward differentiation of cells
INVENTOR(S): Zhong, Yilin; Yan, Ronglang; Wang, Aijun; Yao, Lifen

PATENT ASSIGNEE(S): Peop. Rep. China
SOURCE: Faming Zhuanli Shenqing Gongkai Shuomingshu, 34 pp.
CODEN: CNXXEV
DOCUMENT TYPE: Patent
LANGUAGE: Chinese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
CN 1343489	A	20020410	CN 2000-128685	20000920 <--

PRIORITY APPLN. INFO.: CN 2000-128685 20000920 <--

AB The ointment and intra-oral ointment for treating superficial neoplasm and hyperplasia or untoward differentiation are composed of 0.1-40% phenylbutyric acid or its medical salt, oily ointment substrate, and/or additives (such as antiseptic, thickener, and/or 1,2-propanediol osmotic promotor), and its pH is 4-6. The oily ointment substrate is composed of higher fatty acid (such as stearic acid, oleic acid, myristic acid, or docosanoic acid), wax, grease (such as mineral oil, silicone oil, or vaseline), glycerol, higher alc. (such as sperm oil, stearyl alc., hexadecanol, docosanol, etc), and synthetic lipid.

L45 ANSWER 8 OF 72 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2003:76589 CAPLUS
DOCUMENT NUMBER: 138:131139
TITLE: Cell-cycle drugs for the prevention and treatment of Alzheimer's disease
INVENTOR(S): Nagy, Zsuzsanna
PATENT ASSIGNEE(S): Isis Innovation Limited, UK
SOURCE: PCT Int. Appl., 68 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003007925	A1	20030130	WO 2002-GB3327	20020719 <--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2003032673	A1	20030213	US 2002-200023	20020719 <--
EP 1408938	A1	20040421	EP 2002-749036	20020719 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				

PRIORITY APPLN. INFO.: GB 2001-17645 A 20010719 <--
WO 2002-GB3327 W 20020719

AB The invention relates to therapeutic agents for use in the prevention or treatment of Alzheimer's disease. In particular the invention relates to use of inhibitors of cell cycle re-entry and progression to the G1/S transition or inhibitors of progression of the cell cycle through the G1/S transition point in the prevention or treatment of Alzheimer's disease.

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L45 ANSWER 9 OF 72 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2002:869074 CAPLUS
DOCUMENT NUMBER: 137:363085
TITLE: Treatment of neurodegenerative, psychiatric, and other nervous system disorders associated with polyglutamine expansion using histone deacetylase inhibitors
INVENTOR(S): Steffan, Joan S.; Thompson, Leslie M.; Marsh, J. Lawrence; Bodai, Laszlo; Pallos, Judit
PATENT ASSIGNEE(S): The Regents of the University of California, USA
SOURCE: PCT Int. Appl., 69 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE	
WO 2002090534	A1	20021114	WO 2002-US14167		20020502	<--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW						
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EP 1390491	A1	20040225	EP 2002-769340		20020502	<--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR						
US 2004142859	A1	20040722	US 2003-476627		20031030	
US 2005227915	A1	20051013	US 2004-768292		20040129	<--
PRIORITY APPLN. INFO.:			US 2001-288215P	P	20010502	<--
			US 2002-372724P	P	20020411	
			WO 2002-US14167	W	20020502	
			US 2003-443717P	P	20030129	
			US 2003-476627	A2	20031030	

AB The invention relates to a novel method for treating a variety of diseases and disorders, including polyglutamine expansion diseases such as Huntington's disease, neurol. degeneration, psychiatric disorders, and protein aggregation disorders and diseases, comprising administering to patients in need thereof of a therapeutically effective amount of one or more deacetylase inhibitors. Specifically, histone deacetylases are targeted to limit the consequences of aberrant interaction between polyglutamine expansion variants of proteins and transcription factors, such as p53, to prevent aberrant gene expression. The invention is also directed to a transgenic fly useful as a model of polyglutamine expansion diseases, which may be used to test potential therapeutic agents.
REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L45 ANSWER 10 OF 72 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:658751 CAPLUS
DOCUMENT NUMBER: 137:195535
TITLE: Life extension of Drosophila by a drug treatment
INVENTOR(S): Benzer, Seymour; Min, Kyung-Tai
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 23 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002120008	A1	20020829	US 2001-895141	20010629 <--
PRIORITY APPLN. INFO.:			US 2000-215401P P	20000629 <--

AB The present invention provides methods for extending the life span of a subject and methods for inducing mol. changes within a whole organism that are responsible for the extended life span of the organism; therefore, providing a whole organism system to identify mols. involved in the ageing process. The present invention provides methods for extending the life span of a subject by administering an inhibitor of histone deacetylase (e.g. butyric acid derivative) to the subject, in an amount effective to extend the life, of the subject. In addition, the present invention provides methods for identifying mols. that extend the life span of a subject. This method is carried out by administering to the subject a mol. of interest and an inhibitor of histone deacetylase. Also, the present invention provides methods for identifying mol. alterations in a subject administered an inhibitor of histone deacetylase to induce ageing or extended life span duration. The identification of a mol. alteration in the subject is done by determining the presence, level and/or modification of nucleic acids or proteins in the subject and comparing that with mol. alterations in a subject not administered or exposed to the inhibitor of histone deacetylase.

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=> s composition or formulation
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        312836 COMPOSITIONS
        987542 COMPOSITION
                  (COMPOSITION OR COMPOSITIONS)
       1452617 COMPN
        588915 COMPNS
       1780890 COMPN
                  (COMPN OR COMPNS)
       2240460 COMPOSITION
                  (COMPOSITION OR COMPN)
        143185 FORMULATION
         93975 FORMULATIONS
        208839 FORMULATION
                  (FORMULATION OR FORMULATIONS)
L46    2415400 COMPOSITION OR FORMULATION

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L47         11 L45 AND L46

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L47  ANSWER 1 OF 11  CAPLUS  COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER:         2003:201472  CAPLUS
DOCUMENT NUMBER:          138:210369
TITLE:                    Prolonged-release forms pharmaceutical dosage forms
INVENTOR(S):              Truog, Peter
PATENT ASSIGNEE(S):       Lunamed AG, Switz.
SOURCE:                   Eur. Pat. Appl., 8 pp.
                          CODEN: EPXXDW
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:
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PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 1291015	A1	20030312	EP 2001-810865	20010910 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				

CA 2459165 A1 20030320 CA 2002-2459165 20020904 <--
WO 2003022253 A1 20030320 WO 2002-CH486 20020904 <--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
EP 1427396 A1 20040616 EP 2002-754105 20020904 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK
CN 1553799 A 20041208 CN 2002-817630 20020904 <--
JP 2005508901 T 20050407 JP 2003-526383 20020904 <--
US 2004180962 A1 20040916 US 2004-488276 20040226 <--
PRIORITY APPLN. INFO.: EP 2001-810865 A 20010910 <--
WO 2002-CH486 W 20020904

AB A pharmaceutical unit dosage form comprises a therapeutically ED of a 4-phenylbutyric acid salt having prolonged release of the active ingredient, being suitable for alleviating and curing various diseases upon once or twice daily oral administration. A method for the preparation of the pharmaceutical formulation and the use thereof for the treatment of benign prostate hyperplasia, cancer, leukemias, cystic fibrosis, AIDS, kidney and liver diseases, thalassemia and urea cycle disorders after twice-daily oral administration of the formulation to a patient is also disclosed. A mixture of 6000.0 g sodium 4-phenylbutyrate, 6280.0 g lactose monohydrate, 3500.0 g Methocel K100M, and 750.0 g Avicel PH102, is wetted with 4000.0 g with water, and dried in cold air for 18 h. The mixture is forced through a sieve and dried again for 10 h with air of 40°. A mixture of 240.0 g talcum and 30.0 g magnesium stearate is admixed for 20 min and the mixture is pressed into tablets of 0.70 g each. The cores are provided with a film coating by using a colloidal dispersion containing 7850 g iso-PrOH, 3360 g Eudragit L12.5, 66 g di-Bu phthalate, 18.0 g Miglyol-812 and 56 g PEG-400. The film-coated tablets are dried in a circulating air drying cabinet for at least 4 h at 35°.

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L47 ANSWER 2 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:594666 CAPLUS
DOCUMENT NUMBER: 137:135074
TITLE: Use of retinoids plus histone deacetylase inhibitors to inhibit the growth of solid tumors
INVENTOR(S): Gudas, Lorraine J.; Nanus, David
PATENT ASSIGNEE(S): Cornell Research Foundation, Inc., USA
SOURCE: PCT Int. Appl., 40 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO. KIND DATE APPLICATION NO. DATE
WO 2002060430 A1 20020808 WO 2002-US2976 20020201 <--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ,

UA, UG, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2002183388 A1 20021205 US 2002-61101 20020201 <--
PRIORITY APPLN. INFO.: US 2001-265651P P 20010201 <--

AB The invention provides a method of inhibiting growth of solid tumors in an animal which comprises administering an effective amount of trichostatin A to an animal in need of such treatment. The invention also provides a method of inhibiting growth of solid tumors in an animal which comprises administering an effective amount of a histone deacetylase inhibitor and a retinoid to an animal in need of such treatment. Examples of solid tumors which may be treated using the methods of the invention include but are not limited to carcinomas of the head and neck, breast, skin, kidney, oral cavity, colon, prostate, pancreas and lung.

REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L47 ANSWER 3 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:354085 CAPLUS
DOCUMENT NUMBER: 136:345845
TITLE: Topical aromatic fatty acid composition
INVENTOR(S): Chung, Yih-lin; Yen, Rong-lang; Wang, Ae-june; Yao, Lin-fen
PATENT ASSIGNEE(S): Taiwan
SOURCE: U.S. Pat. Appl. Publ., 8 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002055542	A1	20020509	US 2001-938926	20010824 <--
US 6538030	B2	20030325		

PRIORITY APPLN. INFO.: TW 2000-89119330 A 20000920 <--

AB A method for treating a subject having an ulcer or radiation fibrosis comprises topically administrating to the subject an effective amount of an aromatic fatty acid, its salt or a prodrug. For example, a topical compn. for use in a sustained-release formulation (e.g., a patch) was prepared containing PF-127 2%, Na CM-cellulose 12%, water 82.8523%, Na 4-phenylbutyrate 1.14771%, and 85% phosphoric acid 2%. The compn. form a uniform semisolid, yellow-white in color.

L47 ANSWER 4 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:275750 CAPLUS
DOCUMENT NUMBER: 136:273202
TITLE: Compositions and methods for treatment of cystic fibrosis
INVENTOR(S): Rubenstein, Ronald C.; Reenstra, William
PATENT ASSIGNEE(S): The Children's Hospital of Philadelphia, USA
SOURCE: PCT Int. Appl., 73 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002028348	A2	20020411	WO 2001-US30897	20011004 <--
WO 2002028348	A3	20020613		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2002011379	A5	20020415	AU 2002-11379		20011004 <--
US 2002115619	A1	20020822	US 2001-970843		20011004 <--
PRIORITY APPLN. INFO.:			US 2000-237899P	P	20001004 <--
			WO 2001-US30897	W	20011004 <--

AB The invention includes a method of enhancing the chloride ion transport function of a mutant CFTR polypeptide in epithelial cells in a mammal. In a preferred embodiment, the mammal is a human patient afflicted with cystic fibrosis (CF). Specifically, the method comprises administering to a patient a therapeutically effective amount of a first compound to enhance trafficking of a mutant CFTR polypeptide to the surface of epithelial cells in the patient, and a therapeutically effective amount of a second compound to increase the chloride ion transport activity of a mutant CFTR polypeptide at the surface of epithelial cells, whereby, the chloride ion transport function of the mutant CFTR polypeptide is enhanced. The invention also includes a method of treating CF in a patient, wherein a mutant CFTR polypeptide is present in an epithelial cell in a patient with CF.

L47 ANSWER 5 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER:	2001:935389 CAPLUS
DOCUMENT NUMBER:	136:48419
TITLE:	Use of C4-10 acids for preventing gram-negative bacterial infections
INVENTOR(S):	Popoff, Michel Yvan
PATENT ASSIGNEE(S):	Institut Pasteur, Fr.
SOURCE:	PCT Int. Appl., 32 pp. CODEN: PIXXD2
DOCUMENT TYPE:	Patent
LANGUAGE:	French
FAMILY ACC. NUM. COUNT:	1
PATENT INFORMATION:	

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001097791	A2	20011227	WO 2001-FR1971	20010622 <--
WO 2001097791	A3	20020606		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
FR 2810546	A1	20011228	FR 2000-7992	20000622 <--
FR 2810547	A1	20011228	FR 2000-8383	20000629 <--
FR 2810547	B1	20040130		
CA 2413284	A1	20011227	CA 2001-2413284	20010622 <--
EP 1292291	A2	20030319	EP 2001-947582	20010622 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
US 2004116523	A1	20040617	US 2003-312047	20030207 <--
PRIORITY APPLN. INFO.:			FR 2000-7992 A	20000622 <--
			FR 2000-8383 A	20000629 <--

WO 2001-FR1971 W 20010622 <--

OTHER SOURCE(S): MARPAT 136:48419

AB The invention concerns the use of C4-10 acids and/or at least one of the salts or esters thereof for preparing a pharmaceutical compn. for preventing gram-neg. bacterial infections, in particular Salmonella. Efficacy of caprylic acid in the treatment of guinea pigs infected with Salmonella is described.

L47 ANSWER 6 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2001:885743 CAPLUS

DOCUMENT NUMBER: 136:616

TITLE: Pharmaceutical composition containing a polymer-phenylalkylcarboxylate salt association or conjugate, conjugate polymers, and use in cancer treatment

INVENTOR(S): Avramoglou, Thierry; Bagheri, Rozita; Chaubet, Frederic; Crepin, Michel; Dahri-Correia, Latifa; Dibenedetto, Melanie; Gervelas, Claudia; Huynh, Remi; Jozefonvicz, Jacqueline

PATENT ASSIGNEE(S): Biodex, Fr.

SOURCE: PCT Int. Appl., 50 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: French

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001091742	A1	20011206	WO 2001-FR1672	20010530 <--
W: CA, JP, US				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR				
FR 2809735	A1	20011207	FR 2000-7117	20000602 <--
FR 2809735	B1	20030801		
CA 2410882	A1	20011206	CA 2001-2410882	20010530 <--
EP 1289515	A1	20030312	EP 2001-940634	20010530 <--
EP 1289515	B1	20041013		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY, TR				
JP 2003534374	T	20031118	JP 2001-587758	20010530 <--
AT 279187	T	20041015	AT 2001-940634	20010530 <--
US 2003171333	A1	20030911	US 2003-297035	20030502 <--

PRIORITY APPLN. INFO.: FR 2000-7117 A 20000602 <--
WO 2001-FR1672 W 20010530 <--

OTHER SOURCE(S): MARPAT 136:616

AB The invention discloses a pharmaceutical compn. containing at least one polymer (e.g. dextran) associated or conjugated with at least a phenylalkylcarboxylic acid derivative, polymers conjugated with at least one phenylalkylcarboxylic acid derivative, and their uses in particular in cancer treatment. Conjugate preparation is described.

REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L47 ANSWER 7 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1999:483377 CAPLUS

DOCUMENT NUMBER: 131:125449

TITLE: Transcription therapy for cancers using a retinoic acid and/or an inhibitor of histone deacetylase

INVENTOR(S): Pandolfi, Pier Paolo; Warrell, Raymond P., Jr.; Zelent, Arthur

PATENT ASSIGNEE(S): Sloan Kettering Institute for Cancer Research, USA

SOURCE: PCT Int. Appl., 79 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 9937150	A1	19990729	WO 1999-US1212		19990120 <--
W: CA, JP					
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE					
US 6262116	B1	20010717	US 1998-154672		19980918 <--
PRIORITY APPLN. INFO.:			US 1998-72279P	P	19980123 <--
			US 1998-154672	A	19980918 <--

AB The invention provides a method of treating a neoplastic condition in an individual, comprising administering a pharmacol. ED of a retinoic acid and/or an inhibitor of histone deacetylase. Also provided is a pharmaceutical compn. comprising a retinoic acid, an inhibitor of histone deacetylase, and a pharmaceutically acceptable carrier. Further provided is a method of inducing terminal differentiation of tumor cells in a tumor in an individual in need of such treatment, comprising the step of administering a pharmacol. ED of a retinoic acid and/or an inhibitor of histone deacetylase.

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L47 ANSWER 8 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1999:7813 CAPLUS
DOCUMENT NUMBER: 130:71529
TITLE: Therapeutic nanospheres containing sodium 4-phenylbutyrate for treatment of cystic fibrosis by CFTR gene therapy
INVENTOR(S): Walsh, Scott; Rubenstein, Ronald; Zeitlin, Pamela; Leong, Kam
PATENT ASSIGNEE(S): Johns Hopkins University School of Medicine, USA
SOURCE: PCT Int. Appl., 24 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 9856370	A2	19981217	WO 1998-US11880		19980611 <--
WO 9956370	A3	19990401			
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM					
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG					
CA 2303268	A1	19981217	CA 1998-2303268		19980611 <--
AU 9880624	A	19981230	AU 1998-80624		19980611 <--
AU 749032	B2	20020620			
EP 989849	A2	20000405	EP 1998-928941		19980611 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI					
US 6207195	B1	20010327	US 1998-95882		19980611 <--
JP 2002506436	T	20020226	JP 1999-503069		19980611 <--
PRIORITY APPLN. INFO.:			US 1997-49497P	P	19970613 <--
			WO 1998-US11880	W	19980611 <--

AB 4-Phenylbutyrate exerts many beneficial biol. effects. It appears to induce the transcription of certain promoters, as well as having a remedial effect on proteins which are aberrantly localized within the cell. In addition, it appears to cause cells to developmentally differentiate. The present invention provides nanosphere formulations of 4-phenylbutyrate and other drugs which remediate defective protein localization intracellularly and can be used for treating cystic fibrosis. These formulations permit lower concns. of drugs to be administered, providing both cost and safety benefits.

L47 ANSWER 9 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1997:394840 CAPLUS
DOCUMENT NUMBER: 127:76021
TITLE: Compositions and methods using phenylacetic acid derivatives for therapy and prevention of pathologies, including cancer, AIDS and anemia
INVENTOR(S): Samid, Dvorit
PATENT ASSIGNEE(S): United States Dept. of Health and Human Services, USA
SOURCE: U.S., 61 pp., Cont.-in-part of U.S. Ser. No. 779,774.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5635532	A	19970603	US 1993-135661	19931012 <--
US 6037376	A	20000314	US 1991-779744	19911021 <--
EP 1108427	A2	20010620	EP 2000-126980	19921013 <--
EP 1108427	A3	20040107		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, IE				
EP 1108428	A2	20010620	EP 2000-126981	19921013 <--
EP 1108428	A3	20040107		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, IE				
ES 2171400	T3	20020916	ES 1992-922550	19921013 <--
EP 1484058	A2	20041208	EP 2004-15994	19921013 <--
EP 1484058	A3	20050427		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, IE				
EP 1484059	A2	20041208	EP 2004-15995	19921013 <--
EP 1484059	A3	20050420		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, IE				
ZA 9208140	A	19940421	ZA 1992-8140	19921021 <--
CA 2108963	A1	19950422	CA 1993-2108963	19931021 <--
CA 2108963	C	19990316		
US 5605930	A	19970225	US 1994-207521	19940307 <--
IL 111251	A	20040620	IL 1994-111251	19941011 <--
CA 2173976	A1	19950420	CA 1994-2173976	19941012 <--
WO 9510271	A2	19950420	WO 1994-US11492	19941012 <--
WO 9510271	A3	19950622		
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RW: KE, MW, SD, SZ, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9479737	A	19950504	AU 1994-79737	19941012 <--
AU 702051	B2	19950504		
ZA 9407964	A	19960306	ZA 1994-7964	19941012 <--
EP 725635	A1	19960814	EP 1994-930694	19941012 <--
EP 725635	B1	20041229		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				

JP 09506079	T	19970617	JP 1995-511977	19941012 <--
JP 3628694	B2	20050316		
NZ 275673	A	20000929	NZ 1994-275673	19941012 <--
JP 2001253821	A	20010918	JP 2001-69516	19941012 <--
JP 2003119130	A	20030423	JP 2002-302292	19941012 <--
AT 285760	T	20050115	AT 1994-930694	19941012 <--
EP 1523982	A2	20050420	EP 2004-30912	19941012 <--
EP 1523982	A3	20050427		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT				
PT 725635	T	20050531	PT 1994-930694	19941012 <--
ES 2233931	T3	20050616	ES 1994-930694	19941012 <--
US 5635533	A	19970603	US 1995-470229	19950606 <--
US 5654333	A	19970805	US 1995-465941	19950606 <--
US 5661179	A	19970826	US 1995-469466	19950606 <--
US 5708025	A	19980113	US 1995-465835	19950606 <--
US 5710178	A	19980120	US 1995-469691	19950606 <--
US 5712307	A	19980127	US 1995-465924	19950606 <--
US 5843994	A	19981201	US 1995-478264	19950607 <--
US 5877213	A	19990302	US 1995-484817	19950607 <--
US 5883124	A	19990316	US 1995-484615	19950607 <--
US 5852056	A	19981222	US 1996-633833	19960410 <--
JP 2005139208	A	20050602	JP 2005-54743	20050228 <--
JP 2005139209	A	20050602	JP 2005-54744	20050228 <--
PRIORITY APPLN. INFO.:			US 1991-779744	A2 19911021 <--
			EP 1992-922550	A3 19921013 <--
			US 1993-135661	A2 19931012 <--
			US 1994-207521	A 19940307 <--
			EP 1994-930694	A3 19941012 <--
			JP 1995-511977	A3 19941012 <--
			JP 2001-69516	A3 19941012 <--
			WO 1994-US11492	W 19941012 <--
			EP 2000-126980	A3 20001208 <--
			EP 2000-126981	A3 20001208 <--

OTHER SOURCE(S): MARPAT 127:76021

AB Compns. and methods are disclosed for treating anemia, cancer, AIDS, or severe β-chain hemoglobinopathies by administering a therapeutically effective amount of phenylacetate or pharmaceutically acceptable derivs. thereof or derivs. thereof alone or in combination or in conjunction with other therapeutic agents. Pharmacol.-acceptable salts alone or in combinations and methods of preventing AIDS and malignant conditions, and inducing cell differentiation are also aspects of this invention. Compds. of the invention include R0C(R1)(R2)[C(R3)(R4)]nC(O)OH [R0 = (substituted) Ph, (substituted) naphthyl, (substituted) phenoxy, where the substitution is 1-4 halo moieties, OH, lower straight-chain or branched alkyl; R1, R2 = H, OH, lower alkoxy, halo, lower straight-chain or branched alkyl; R3, R4 = H, lower alkoxy, halo, lower straight-chain or branched alkyl; n = 0-2] and pharmaceutically acceptable salts and mixts. thereof.

L47 ANSWER 10 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1997:196180 CAPLUS
DOCUMENT NUMBER: 126:207539
TITLE: Compositions and methods using phenylacetate compounds, alone or in combination with other therapeutic agents, for treating and preventing anemia, cancer, and other pathologies and modulating lipid metabolism
INVENTOR(S): Samid, Dvorit
PATENT ASSIGNEE(S): United States Dept. of Health and Human Services, USA
SOURCE: U.S., 111 pp., Cont.-in-part of U.S. Ser. No. 135,661.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English

FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
US 5605930	A	19970225	US 1994-207521	19940307	<--
US 6037376	A	20000314	US 1991-779744	19911021	<--
EP 1108427	A2	20010620	EP 2000-126980	19921013	<--
EP 1108427	A3	20040107			
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, IE					
EP 1108428	A2	20010620	EP 2000-126981	19921013	<--
EP 1108428	A3	20040107			
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, IE					
EP 1484058	A2	20041208	EP 2004-15994	19921013	<--
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EP 1484059	A2	20041208	EP 2004-15995	19921013	<--
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IL 111251	A	20040620	IL 1994-111251	19941011	<--
CA 2173976	A1	19950420	CA 1994-2173976	19941012	<--
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AU 9479737	A	19950504	AU 1994-79737	19941012	<--
AU 702051	B2	19950504			
EP 725635	A1	19960814	EP 1994-930694	19941012	<--
EP 725635	B1	20041229			
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JP 09506079	T	19970617	JP 1995-511977	19941012	<--
JP 3628694	B2	20050316			
NZ 275673	A	20000929	NZ 1994-275673	19941012	<--
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JP 2003119130	A	20030423	JP 2002-302292	19941012	<--
AT 285760	T	20050115	AT 1994-930694	19941012	<--
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PT 725635	T	20050531	PT 1994-930694	19941012	<--
ES 2233931	T3	20050616	ES 1994-930694	19941012	<--
US 5843994	A	19981201	US 1995-478264	19950607	<--
US 5883124	A	19990316	US 1995-484615	19950607	<--
US 5852056	A	19981222	US 1996-633833	19960410	<--
JP 2005139208	A	20050602	JP 2005-54743	20050228	<--
JP 2005139209	A	20050602	JP 2005-54744	20050228	<--

PRIORITY APPLN. INFO.:	US 1991-779744	A2	19911021	<--
	US 1993-135661	A2	19931012	<--
	EP 1992-922550	A3	19921013	<--
	US 1994-207521	A	19940307	<--
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	JP 1995-511977	A3	19941012	<--
	JP 2001-69516	A3	19941012	<--
	WO 1994-US11492	W	19941012	<--
	EP 2000-126980	A3	20001208	<--
	EP 2000-126981	A3	20001208	<--

OTHER SOURCE(S): MARPAT 126:207539

AB Compns. and methods are disclosed for treating anemia, cancer, AIDS, or severe β-chain hemoglobinopathies by administering a therapeutically effective amount of phenylacetate or (pharmaceutically acceptable) derivs. thereof alone or in combination or in conjunction with other therapeutic agents including retinoids, hydroxyurea, and flavonoids. Also disclosed are intravesical methods of treatment of cancers with phenylacetate. Pharmacol.-acceptable salts alone or in combination, and methods of preventing AIDS and malignant conditions and inducing cell differentiation are also aspects of this invention. A product as a combined preparation of phenylacetate and a retinoid, hydroxyurea, or flavonoid (or other mevalonate pathway inhibitor) is disclosed for simultaneous, sep., or sequential use in treating a neoplastic condition in a subject. Also disclosed are methods of modulating lipid metabolism and/or reducing serum triglycerides in a subject using phenylacetate.

L47 ANSWER 11 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1995:638522 CAPLUS
DOCUMENT NUMBER: 123:25666
TITLE: Phenylacetate and derivatives alone or in combination with other compounds against neoplastic conditions and other disorders
INVENTOR(S): Samid, Dvorit
PATENT ASSIGNEE(S): United States Dept. of Health and Human Services, USA
SOURCE: PCT Int. Appl., 182 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9510271	A2	19950420	WO 1994-US11492	19941012 <--
WO 9510271	A3	19950622		
W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, JP, KE, KG, KP, KR, KZ, LK, LR, LT, LU, LV, MD, MG, MN, MW, NL, NO, NZ, PL, PT, RO, RU, SD, SE, SI, SK, TJ, TT, UA, US, UZ				
RW: KE, MW, SD, SZ, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
US 5635532	A	19970603	US 1993-135661	19931012 <--
US 5605930	A	19970225	US 1994-207521	19940307 <--
AU 9479737	A	19950504	AU 1994-79737	19941012 <--
AU 702051	B2	19950504		
EP 725635	A1	19960814	EP 1994-930694	19941012 <--
EP 725635	B1	20041229		
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AT 285760	T	20050115	AT 1994-930694	19941012 <--
US 5852056	A	19981222	US 1996-633833	19960410 <--

PRIORITY APPLN. INFO.:

US 1993-135661	A	19931012 <--
US 1994-207521	A	19940307 <--
US 1991-779744	A2	19911021 <--
WO 1994-US11492	W	19941012 <--

OTHER SOURCE(S): MARPAT 123:25666
AB Compns. and methods of treating various disorders by administering a therapeutically effective amount of phenylacetate or pharmaceutically acceptable derivs. thereof or derivs. thereof alone or in combination or in conjunction with other therapeutic agents including retinoids, hydroxyurea, and flavonoids. Intravesicle methods of treatment of cancers phenylacetate. Pharmacol.-acceptable salts alone or in

combinations and methods of preventing AIDS and malignant conditions, and inducing cell differentiation are also aspects of this invention. A product as a combined preparation of phenylacetate and retinoid, hydroxyurea, or flavonoid (or other mevalonate pathway inhibitor) for simultaneous, sep., or sequential use in treating a neoplastic condition in a subject. Methods of modulating lipid metabolism and/or reducing serum triglycerides in a subject using phenylacetate.